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The Effects of Noise on Health

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In recent years, the possibility that noise has a direct adverse effect on the health of people has been the subject of considerable controversy at both public and scientific meetings. Different viewpoints have been expressed in both popular and scientific articles. So many different effects have been claimed that it is almost impossible to address all of the concerns. Reviews in which all or most claims are discussed become very disjointed because of the broad range of topics. Every effect seems to come under the rubric of an health effect, whether it involves interference with some human activity or whether it involves Ischaemic Heart Disease. This probably came about because "Public Health and Welfare" has been mentioned so frequently in articles, regulations and international congresses on noise. This is good in that it is unlikely that any area of effects will be ignored. It is limiting because it can be very difficult, and oftentimes meaningless, to compare effects. For example, the threshold of effects for annoyance are very different from that for cardiovascular disease. Similarly, it is not very easy to separate out the factors that pertain strictly to health from those that pertain to public welfare or quality of life. It is also not very easy to define health or welfare or quality of life separately. For thses reasons, after some experts opinion on the health effects of noise, there is an attempt to convey the complexity of and the difficulties in forming a perspective on noise research. This is followed, in turn, by a discussion of the some of the early research sponsored by the Environmental Protection Agency (EPA) that has had such a large role in developing many of the methods and procedures for noise assessment and evaluation. Many of the methods and recommendations are still used both national and internationally. This is followed by a discussion of noise induced physiological changes, noise as a stress, and on some specialized topics on the effects of impulsive acoustic stimuli, on effects on sleep, and on hearing and health effects of people living under military training routes. Finally, an attempt is made to summarize, evaluate and take a position in the last section.

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Introduction

In recent years, the possibility that noise has a direct adverse effect on the health of people has been the subject of considerable research, received much attention in the popular press, and been the source of considerable controversy at both public and scientific meetings. Different viewpoints have been expressed in both popular and scientific articles. So many different effects have been claimed that it is almost impossible to address all of the concerns. Reviews in which all or most claims are discussed become very disjointed because of the broad range of topics. Every effect seems to come under the rubric of an health effect, whether it involves interference with some human activity or whether it involves Ischaemic Heart Disease. This probably came about because **"Public Health and Welfare"** has been mentioned so frequently in articles, regulations and international congresses on noise. This is good in that it is unlikely that any area of effects will be ignored. It is limiting because it can be very difficult, and oftentimes meaningless, to compare effects. For example, the threshold of effects for annoyance are very different from that for cardiovascular disease. Similarly, it is not very easy to separate out the factors that pertain strictly to health from those that pertain to public welfare or quality of life. It is also not very easy to define health or welfare or quality of life separately. For these reasons, after some experts opinion on the health effects of noise, there is an attempt to convey the complexity of and the difficulties in forming a perspective on noise research. This is followed, in turn, by a discussion of the some of the early research sponsored by the Environmental Protection Agency (EPA) that has had such a large role in developing many of the methods and procedures for noise assessment and evaluation. Many of the methods and recommendations are still used both national and internationally. This is followed by a discussion of noise induced physiological changes, noise as a stress, and on some specialized topics on the effects of impulsive acoustic stimuli, on effects on sleep, and on hearing and health effects of people living under military training routes. Finally, an attempt is made to summarize, evaluate and take a position in the last section.

Opinions of the Experts

There seems little doubt that noise under some conditions can have an effect on the health of people. The real problem is in determining the level at which we may expect noise, as one of many stresses, to be a contributor to disease. Investigators in most, but not all, studies have studied the relationship between some aspect of cardiovascular disease and noise exposure. As we shall see from the opinions of the experts, the results have been inconsistent.

Several years ago, in the EPA Criteria Document, it was pointed out that " Noise can elicit many different physiological responses. However, no clear evidence exists indicating that the continued activation of these responses leads to irreversible changes and permanent health effects (68)." Similarly, Burns after reviewing the difficulties in doing research in this area goes a step even farther by pointing out: "In field studies of real human situations, adequately controlled conditions may be unattainable, so that results deriving from some other factor in the total environment of the subjects may be incorrectly attributed to noise (6)." Kryter, in an early detailed study of the literature, concluded:

> "In spite of the very large gaps in our knowledge and the existence of some apparently conflicting results, the following conclusions are put forth, with, of course, the usual admonition that more research is needed before they can be accepted with great confidence.
>
> (1) There is no likely damage risk to a person from the possible unconditioned stress responses to noise that are mediated by the autonomic system.
> (2) Noise may often be concomitant with danger and adverse social-environmental factors that are more important than the noise itself as a cause of apparent great incidences of various physical and psychological disease and accidents in industry.
> (3) Autonomic system stress responses could conceivably be a contributing factor to ill health in some persons as the result of noise in their living environment directly interfering with auditory communications and sleep, and, thereby, creating the feelings of annoyance and anger that can serve as the direct cause of the stress responses (46)."

Kryters' conclusions are now over twenty years old, however, they are just as appropriate and true today as they were then.

The Committee on Hearing, Bioacoustics, and Biomechanics (CHABA) was asked by the National Institute for Occupational Safety and Health (NIOSH) to recommend research that could be conducted to examine the effects of noise on human health. Specifically, in addition to examining the literature, they were asked whether—"noise standards established to safeguard hearing are sufficient also to protect against health disorders other than hearing deficits."

In their review of a cross-section of the literature, the CHABA Committee (Working Group 81) reports:

> "Additional research will be necessary in order to obtain critical evidence as to whether detrimental health effects, other than those to the auditory system, do or do not occur as the result of long-term exposure to high-level sound. Additional research will also be necessary to determine whether noise standards established for safeguarding hearing are also adequate to minimize any nonauditory health effects from such exposures. The appropriate studies will be difficult and expensive both in cost and in commitment of time by research personnel for data acquisition and analysis. To demonstrate that long-term exposure to high-level noise per se is a risk factor for cardiovascular or other disorders, it is necessary that highly sophisticated epidemiological studies be conducted with controls for other known risk factors. Other risk factors that should be considered are: age, sex, smoking, caffeine, body weight, diet, and hereditary proclivity; factors related to stress associated with physical hazards in job performance and work output requirements; factors in the work environment that might be associated with noise, such as heat, dust, toxic fumes, and vibration; and factors in non-work living environment of the workers. Less careful and complete research will only produce results that are ambiguous as those now available. If new studies are proposed, we recommend that the designs be reviewed by an appropriately qualified group of experts (81)."

Although WG 81 was reluctant to state that protecting against hearing loss was also protecting against any other health effects, an earlier CHABA WG (No.69) was not so reluctant to state this in the "Guidelines" document. In that document a procedure was given for calculating potential

hearing loss and it was also suggested that the procedure can be used for the calculation of the beginnings of physiological effects. Many investigator still believe that protecting against hearing loss is also protecting against other health effects. Von Gierke and Eldred stated:

> "—most authoritative reviews such as the World Health Organization Environmental Criteria Document on noise agree 'research on this subject has not yielded any positive evidence, so far, that disease is caused or aggravated by noise exposure, insufficient to cause hearing impairment.' For practical noise control considerations, the present status of our knowledge means that the criteria for evaluating environmental noise impact, with respect to its direct and indirect effects on health, are the same criteria as those applied to prevent any hearing impairment, and to reduce any annoyance stress to a minimum (93)."

Much of the research that has been conducted was on the effects of noise that occurs in the work place and the effects on workers who have been exposed to very high levels of noise, often more than that allowed currently in the US, for long periods of time, in some cases more than 40 years of exposure. Also, it will be recognized that none of the preceding authors recommend types or levels of noise that must be avoided and certainly no equations are provided for linking noise with health effects such as we now have for linking noise exposure and annoyance and hearing loss. However, one CHABA Working Group did specify a limiting level of noise. The Air Force, with endorsements from NIOSH and the EPA, asked Working Group 85 three questions concerning the effects of high intensity noise on the embryos or fetuses of pregnant women. These questions were: (1) What are the potential hazards of noise exposure? (2) On the basis of current knowledge can reasonable limits be specified for conditions of noise exposure? and (3) What research efforts, if any, should be encouraged to obtain better answers to the first two questions? The committee responded that based on the available evidence they could not give definitive answers to the first two questions, but they did make recommendations for further research. Later in their summary and conclusions section, they state: **"There is no conclusive evidence of detrimental effects of high-intensity external sound in higher mammals."** And that **"Until better information is available—it would appear prudent to avoid exposures of long duration (several hours per day) to sounds of 90 dB SPL and above,** the maximum level currently suggested by the US. Department of Labor for unprotected ears (67)." Later investigations seem to have produced results which suggest more adverse effects of noise on the later hearing of the embryo than on the health of the mother. Although, some studies suggest that noise may produce hypertension in the mother.

The Health Council of the Netherlands was asked by the minister of Welfare, Health and Culture to prepare an advisory report on the effects of noise on human health. The Health Council responded with quite an extensive review by W. Passschier-Vermeer on "Noise and Health" (58). A Health Council committee was then formed and with the report in hand were asked among other factors to : "--estimate the size of the population groups exposed to noise in the Netherlands and the health effects expected in these people." It was pointed out that this committee was an international one and their report represents the state of the art in this area (29). They looked at hearing loss, sleep-disturbances, psycho-social effects including annoyance, the effects on human performance, and stress-related health effects such as hypertension, cardiovascular disease and birthweight.

The evaluation of the health effects for a causal link with noise was patterned after that used by the International Agency for Research on Cancer and by a set of guidelines recommended for

evaluating epidemiological studies. Results of studies were rated using four categories: sufficient evidence of a causal relationship, limited evidence, inadequate evidence, and evidence suggesting a lack of causality. In addition to causality, the committee assigned noise level values where there was **sufficient** evidence of an effect to indicate an **observation threshold** or beginning of health effects.

In some cases an observation threshold was not given because no data were available at lower levels. For example, an effect was obtained at 85 dB but there may have been an effect at a lower value so <85 was entered in the table indicating the effect begins lower than 85 dB. Several difference measures of noise exposure were used and with the exception of some sleep-disturbance effects most were a variation of L_{eq}. The authors point out that it was not possible to use just one measure such as an $L_{eq,24h}$ because of the many different situations necessitating specification of noise source. The only noise measure that may be confusing is $L_{EX,occ}$. This measure refers to an $L_{eq,8h}$ during a representative working day.

Tables 1 and 2 presented below are taken from Table 1 in the committee report (29). Table 1 contains those effects where the evidence was rated sufficient and, consequently, there are accompanying values indicating thresholds of effect. Table 2 contains those effects where the evidence was rated lower than sufficient thus no noise exposure values are given.. The authors point out that the measures used in the table are not necessarily those that they would recommend for use in regulations.

There are few places in the noise literature where investigators or committees were willing to set values for the beginning of health effects. One place was in the Guidelines article (21) where it was pointed out that an $L_{eq,8h}$ of 75 dB(A) (or approximately an $L_{eq,24hr}$ of 70 dB(A)) represented the beginning of potential hearing loss (see Appendix A). And it has been pointed out that it can serve as a surrogate for the beginnings of physiological effects as well. In Table 1 it can be seen that a number of effects begin to occur at 70 dB or lower. These values cannot be compared directly with the 70 dB in the Guidelines article without translating the values to a 24-hour baseline. This can be done by taking 10log(effect time in hours/24 hr). For example, in Table 1 for hypertension, environment road traffic (env road), there is an L_{eq} that includes the hours from 0600 to 2200 a period of 16 hours thus 10log(16/24) is -1.76 which gives 68.24. Similarly, for 85 dB(A) for 8 hours would be log10(8/24) is -4.77 which gives 80.23 dB(A).

Table 1. (Possible) long term effects of exposure to noise, classification of the evidence for a causal relationship and data on the observation threshold (sufficient causality)(ref. 29).

effect	classification of evidence	situation	observation measure	threshold value in dB(A)	in/out
hearing loss	sufficient	occ	$L_{EX,occ}$	75	in
		env recr	$L_{eq,24h}$	70	in
		occ unb	$L_{EX,occ}$	<85	in
hypertension	sufficient	occ ind	$L_{EX,occ}$	<85	in
		env road	$L_{eq,06\text{-}22h}$	70	out
		env air	$L_{eq,06\text{-}22h}$	70	out
Ischaemic heart disease	sufficient	env road	$L_{eq,06\text{-}22h}$	70	out
		env air	$L_{eq,06\text{-}22h}$	70	out
annoyance	sufficient	occ off	$L_{EX,occ}$	<55	in
		occ ind	$L_{EX,occ}$	<85	in
		occ indtraf	L_{dn}	42	out
sleep disturbance, changes in:					
sleep patterns	sufficient	sleep			
awakening	sufficient	sleep	SEL	60	in
sleep stages	sufficient	sleep	SEL	35	in
subjective sleep quality	sufficient	sleep	$L_{eq,night}$	40	out
heart rate	sufficient	sleep	SEL	40	in
mood the next day	sufficient	sleep	$L_{eq,night}$	<60	out
performance	sufficient	school	$L_{eq,school}$	70	out

Table 2. (Possible) long term effects with less than a sufficient rating for causal effect (ref. 29).

effect	classification of evidence	situation
biochemical effects	limited	occ
		env
immune effects	limited	occ
		env
birthweight	limited	occ
		env air
congenital effects	lack	occ
		env
psychiatric disorders	limited	env air
absentee rate	limited	occ ind
		occ off
sleep disturbance, changes in		
hormones	limited	sleep
immune system	inadequate	sleep
performance the next day	limited	sleep
performance	limited	occ env
psycho-social well-being	limited	env

Table 1 perhaps should be broken down further because the sufficient effects for hearing loss, hypertension and ischaemic heart disease do not seem to fit into the same category as annoyance,

sleep disturbance, and human performance. The former seem more strongly related to health. The evidence of a cardiovascular effect seems much more serious. The effect begins at 70 dB(A), however, since we have no dose response relationship it may not be. In any case the U. S. Air Force uses different dose response equations for assessing annoyance and sleep interference (behavioral awakening) than do these European investigators and this makes comparisons difficult. However, there seems little to disagree with on these effects. There is reason to debate about some of the effects listed under hearing loss and cardiovascular disease. First, the interest in the effect of noise on the unborn has shifted from many of the dire consequences (abortion of fetus, cardiovascular disease, etc.) of past years to focus mainly on the possibility that noise exposure of pregnant women may cause permanent threshold shifts in the unborn child. This is based on only two studies and perhaps should be classified under limited evidence. The only two effects that deal directly with aircraft noise are the sufficient causal effects claimed for Hypertension and for Ischaemic heart disease. The authors state:

> "there is little evidence for an increased risk of hypertension and of ischaemic heart disease in people living in areas with traffic noise at outdoor equivalent sound levels (from 06 to 22 hours) below 70 dB(A).
> "the relative risk of ischaemic heart disease and of hypertension starts to increase for persons living in areas with road or air traffic noise at equivalent sound levels above 70 dB(A) (from 06 to 22 hours) (29)."

The studies referred to in the quote have been discussed in the present paper under cardiovascular effects. Nevertheless, the percent highly annoyed is getting to be a problem for many of the people since roughly 20% of the people living in the area would be highly annoyed (see Appendix B).
These experts start off their Table of effects with (possible) effects. One would assume that the **possible** means these thresholds are flexible and subject to change. If this is the case, I would suggest that most values are too conservative as would many of my colleagues and there would be little to disagree with. Nevertheless, it seems a little premature to try to set the beginning of effects in the cardiovascular effects area since the threshold of effects is generally the most difficult to determine. Without a dose-response relationship for an effect, the threshold has little meaning. Usually one starts in an experimental area by looking at the extremes of the experimental variables, which is impossible to do today in the noise effects area because of regulations, rules and laws. In spite of this, there seems reason to disagree with the classification of the evidence in the cardiovascular area as **sufficient.** It seems **limited** at best.

In comparing the results of the present effort in the Netherlands with the 1971 Health Council report, the authors point out that more data is available and more is known about noise effects than two or three decades ago. One would agree that more is known about annoyance reactions, speech communication, and hearing loss but do we really know more about cardiovascular effects. There are some interesting theories. The effects are biologically plausible but have they been proven? The authors conclude:

> "Most other effects of noise, among which ischaemic heart diseases and hypertension, occur at high noise levels: only people carrying out very noisy occupational activities or being in situations with very specific environmental noise exposures may suffer from such effects. –Many of these effects might be prevented by strict enforcement of existing regulatory limits (29)."

Developing a Perspective on the Effects of Noise on Human Health

While there have been a few findings that could be interpreted to mean that noise has an adverse effect on human health, either from surveys or from animal experimentation, the general consensus is that it has not been firmly established that long-term exposure to noise has a direct adverse effect on human health, aside from damage to the auditory system. However, debates among scientists sometime go beyond a discussion of the data to a more abstracted level and the debate may become polarized into a small group of believers and a small group of unbelievers. Most stay somewhere in the middle away from the extremes, nevertheless, there are scientists who believe or think that noise has an adverse effect on health and of course there are those who think it has no effect at all (at least at the levels of current noise exposures). Some, on both sides, even take up their side's position as a cause. They become quite eager to verbalize or write about their position for the press or for scientific meetings or for that manner for any one who will listen. For the sake of discussion, take two scientists, one who believes that noise has adverse effect on health and one who believes it has no effect, which do you think would get more press coverage. Which one would make the most interesting public speaker? Can you imagine a headline "noise has no effect on health" or "noise will not drive you crazy" or "noise is an irritating but innocuous accompaniment of modern life" ? These questions have an easy obvious answer but you can see that the scientist that believes noise has no effect on human health is likely to be at a disadvantage. Not only will the press listen only to the first scientist but they will in all probability over dramatize his actual statements. Therefore, there is a real problem in informing the public as to the position that most scientists have on the subject . It seems to be the nature of things that effects in this area tend to be exaggerated, as witnessed by some of the early articles on the adverse effects of infrasound and ultrasound (28). Therefore, it is not only in the popular press that effects are exaggerated. The EPA put out a pamphlet some years ago, that is still seen today, for public use that several scientists at various meetings have denounced as exaggerating the effects of noise on health. Furthermore, most proposals for research, and other articles as well, on the health effects of noise have bias built into them that is to varying degrees pro-effect. They are more likely to quote studies and discuss the previous literature that proports to show adverse affects of noise, and to either ignore those without "suggestive effects" or to give them only brief passing reference (or sometimes to point out their inadequacies in study design). This is natural and should be expected. Studies that show "effects" provide the hypotheses and the meat for research proposals; nevertheless, one should not expect to get an uncritical review of the state of knowledge in the noise effects area. A critical reader will also recognize that a good deal of over dramatizing and promoting is going on in studies that are submitted either because of the hope of funding or to point out the "critical need" for more research in their area of expertise. Oftentimes, these articles prove guilt by association. The scenario usually goes somewhat like the following: "strokes and heart attacks are the number one killer in the land; noise may increase blood pressure has been shown by so and so, blood pressure is an indicator of hypertension, and it is well known that hypertension is the cause of strokes and heart attacks." Thus, noise often stands indicted on the most meager of

evidence. These remarks are facetious and oversimplified. There is no intention of being facetious about the potential seriousness of the problem or even about the studies that show suggestive effects, but rather about the manner in which some people interpret the findings.

Administrators and Scientists often, in arguing their position, say "the data clearly show" that such and such effect occurred. In research, the data never clearly show anything, they are interpreted logically, statistically, and in the frame of reference of previous research to support or not support some hypothesis or hypotheses. And the results of one study or often a series of studies must be evaluated in the context of all of the rest of the research that has been done in the area. The point is that one cannot pick out one study or a series of studies to support a position. They must consider all studies that have been done and then decisions must be made on the balance of the evidence. This must be said because studies in this area which seem to show adverse effects are quoted out of all reference to their importance, even when the original authors have cautioned against such an interpretation and in many cases would be astonished as to the interpretation other people have put on their results. The facts remain that some studies are quoted over and over, even when evidence exists that seem to indicate the contrary. This is interesting as a parallel to the study of human psychology, which to oversimplify says that once a person has made up his mind about something then he will have a hard time changing it even when he is presented with direct evidence that his first opinion or conclusion no longer accords with the facts. To put it in more psychological language and quote from Janis and Rodin (37) "the perseveration effect apparently occurs because evidence, once coded, becomes autonomous from the coding scheme so that its influence no longer depend on the validity of that scheme."

Some people have for many years taken a broad view of what should be considered "Health Effects." Now many more investigators and reviewers are taking the broader view. There are now more discussions of annoyance, interference with the learning of children, admissions to mental hospitals and many other factors that seem more related to quality of life issues. Most of these effects, if they exist (as a health issue) at all, are based on the most tenuous of evidence. This broadening of the definition is in some ways unfortunate mainly because the field needs focusing not broadening. First, provide one clear-cut example of how noise actually contributes to cardiovascular disease. This, of course, should be a study in which the investigator is aware that a physiological change is not demonstration of a health effect and that a general idea of the noise level is not enough. After we have such a study, we can then proceed to examine health effects in other areas.

Decibel levels that represent the beginnings of adverse health effects in some areas are now specified.. This was what the committee (29) in the Netherlands was asked to do. To the credit of the committee, they in many areas of noise effects pointed out that the evidence was not "sufficient" to choose a threshold. Nevertheless, there seems very little hard evidence for the levels they did specify. They point out that an Leq of 70 based on exposure from 0600 to 2200 was the threshold for hypertension and for ischaemic heart disease. It didn't matter if the noise was road traffic or aircraft noise. This is truly puzzling considering the road traffic study they quoted yielded mostly nonsignificant results at this level except the highly annoyed in one case had a higher risk ratio for cardiovascular disease. This level does show some agreement with a level specified as the beginnings of possible physiological effects in the Guidelines document (21) published some years earlier.

It is also difficult to understand the concept of the lowest level (threshold) where one starts to get an effect. What does this mean and how serious is it? Are is it serious for some people and not for others? In the Levels document, up to 70 dB 24 hour LEQ is suggested as safe for human hearing. This is the level where after 40 years of exposure would produce less than 5 dB Noise Induced Permanent Threshold Shift (NIPTS) in 100% of the people at the most sensitive frequency of 4000 kHz (32). This same level is used in the Guidelines document (21) for beginning the calculation of NIPTS, where it has also been suggested that one can begin the calculation of physiological effects.

It is sometimes a puzzlement how some people reach conclusions regarding the effects of noise on human health. Fortunately, there is a quote used by Lipscomb (49) which says something to the effect that "it is easy to overestimate how much the people know and easy to underestimate their intelligence." Most people can recognize hype whether it is in advertising or in pronouncements about the adverse effects of noise on human health.

In the area of the effects of noise on health, contrary to common sense, it is probably better that any pronouncements about the effects of noise come from a committee. Certainly, it will require a team of experts to conduct good research in this area because of the many diverse problems of methodology, and experimental and statistical control.

Complexity of the Problem

All surveys of the literature on the effects of noise on human health emphasize the immense complexities involved in determining whether there is a relationship or not. In spite of this, the complexity of the problem is even greater, in most cases, than has been previously portrayed. This complexity can be emphasized by paraphrasing from Stone (78). A system may be considered to be a collection of entities interacting toward some purpose. In the present case, the entities can be considered to be independent variables (those that cause the effect) and the purpose would be to determine an effect on health which we will define as an increase in blood pressure (our dependent variable gives evidence of the effect). Noise, of course, is one of our independent variables and we must choose other variables besides noise that are likely to have an effect on blood pressure. To determine the other independent variables, some previously acquired data or a good theory is needed to guide us. Once we have some idea of the independent variables (elements) that must be included in the system, we have to recognize that each element has multiple states or levels. Noise has an infinite number of levels, and of course, one cannot assume that each level has a predictable effect; in some cases different levels may have opposite effects. It is usually more complicated than this because most elements of a system are related to more than one, usually to many other elements of the system. What this means is that if there is a change in element A of the system from level 1 to level 2, this increases the likelihood that element B will change from level 2 to the level 3 and maybe that

element C will change from level 6 to level 5, etc. Therefore, there would not be one relationship between noise level and blood pressure but a whole family of relationships; all depending on the number of elements and the number of states of the elements that are in the system. A direct quote from Stone should serve to illustrate this complexity:

> "For any but the most trivial systems, a complete description is an unattainable ideal. For example, if we were concerned with a system of three elements, each taking on only three states and assuming that the system had no memory of how it got to its present state—a one-stage Markovian System—we would need 648 simple rules to specify all of the relationships—If we had four elements, we would require 27,648 simple rules—(78)."

In the study of risk factors (independent variables or elements) in cardiovascular research, there are many more than four risk factors that must be considered. There are more than that have been demonstrated to bear at least some relationship to cardiovascular disease. In fact, in the cardiovascular area, there are families of variables that must be considered. There are those related to the psychological, sociological, and biological nature of man. For an example of psychological variables consider, as have many psychologists and cardiologists, that personality traits may predispose some people to certain emotional states that thereby affect their appraisals and subsequent emotion reactions to various situations whether they be noise exposure, crowding, odors or heat. These reactions can, in turn, produce inadequate coping strategies for dealing with the situation (such as smoking, drinking or drug abuse) which lead to impaired health or more indirectly may lead to anxiety or to lowered self-esteem so the person sees himself as unable to cope and becomes depressed and this also may affect health.

The problem in studying the effects of noise on human health, in particular on cardiovascular function, is that no one has been able to demonstrate a direct cause and effect relationship. Many surveys of workers who have been exposed to high levels of noise, often more than 90 dBA, for many years have, of necessity, reported their results in terms of correlation. Correlation, as Statisticians are fond of saying, does not indicate a cause and effect relationship. Some surveys have shown that workers in some industries have higher blood pressure than the general population (no study has been conducted, however, without methodological flaws). They have not shown, however, that noise produced the higher blood pressure. The high blood pressure could have been caused by stresses of the job, time pressure to work rapidly or danger of moving equipment, or other environmental factors such as dust, heat or cold, odors, or vibration. To quote from Johnson:

> "—One can reasonably assert that noise is a by-product of those kinds of jobs that probably do cause more stress. Remember, we can demonstrate that noise can cause permanent changes to the ear; but we have no similar data for blood pressure. Thus we can only make a conjecture that there might be a cause and effect relation. Such a relation might be shown if we could find two groups of people identical in all ways except for noise exposure. Until such proof is forthcoming, I believe that such possible effects must be ignored in the current planning or decision making process (40)."

This is essentially the same position recommended by von Gierke and Eldred in a more recent review of the effects of noise on people (93).

Burns (6) has reviewed the literature on the effects of noise on human physiology. He discussed the short-term effects and the long-term effects, the possible effects as indexed by changes in blood pressure, pulse, peripheral constriction of blood vessels, muscle tension, the startle response, eye pupil responses, gastrointestinal motility, and chemical and hormonal changes in

the body. Also, he reviewed the studies in which animals have been used to evaluate the effects of noise. In view of the many types of essentially diverse approaches to the study of the adverse effects of noise, his conclusions are quite interesting. He states:

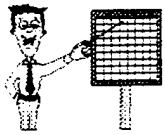
> "The epidemiological study of industrial absenteeism, accident rates, injury rates, and any other possible index (such as fetal abnormalities) against the background of the occupational environment including noise must, despite its daunting difficulties, be the principle means of access to direct evidence of the effects, if any, of noise exposure of a general nature. The difficulties of separating noise from other environmental factors have been faced in one study. Unfortunately no firm conclusion is possible in this area at present; caution, vigilance, and an open mind on the possibilities of noise effects must underlie future work. In view of the inconclusive state of the evidence, regulatory restriction of occupational noise exposure in order to protect hearing is also likely to restrict other possible effects (6)."

The interesting aspect of this conclusion is that he has recommended probably the most complex situation possible be used (researched) to attempt to find a link between noise exposure and health (as well as adverse effects on behavior). This conclusion seems to be the most reasonable one that can be reached at the present time. There are numerous reasons why this is so. One is that in the occupational environments, even with the current legislative restrictions on the permissible noise levels, people are exposed to higher noise levels for longer periods of time than they are in any other situation (as measured by energy-averaging techniques such as Leq). (However, these levels are not greater than some people experience voluntarily in their leisure time activities, i.e., rock concerts, high powered car player and radio, motorcycle riding, using chain saws, etc.). Since there has been great difficulty in establishing a cause and effect relationship between noise and any health effect, it seems most reasonable to look for such effects in those situations where people are exposed to the most intense noise. Furthermore, the complexities involved in studying noise in the workplace may not be any more involved than the problem of generalizing from the results of studies where human subjects are exposed to brief periods of intense noise or from studies in which animals are exposed to noise. However, Schwarze and Thompson caution:

> "On the basis of current and past research, it seems unlikely that studies in industries where adequate noise protection is maintained will show very strong effects of noise on the cardiovascular system. Prospective studies in work environments with currently permissible noise levels and sustained use of hearing protectors are needed to clarify the conditions under which noise exposure may adversely affect the cardiovascular system. (72)."

Noise may not only be a weak variable under such circumstances but it will probably meet the same fate that some have suggested for hearing loss studies. That with modern regulations the time may have past to do good studies. There simply isn't any loss that may be attributed to noise exposure. This is probably not the case in either hearing loss or health studies, but we are going to have to know, as Thompson (86) has pointed out, the total noise environment of the individual. It is only in this way that noise level can truly be related to health effect measures. And even though the noise exposure is regulated, it is still likely to be the place where most people receive the most noise.

Early EPA Sponsored Research



Although, EPA-sponsored research in the noise area is minimal today, that was not the case in the 1970s and early 1980s. It is necessary and important to examine the decisions and recommendations that EPA (mainly through committees and sponsored research) has made. Their positions have become the prevailing ones for preparing Environmental Impact Statements (ElSs) and they impact directly on most decisions made about noise in the USA and in many places throughout the world. The information contained in the Criteria Document, the Levels Document, the CHABA Guidelines, and the report of CHABA Working Group 81 on impulse noise is important today. Many conferences have gradually expanded but mostly reviewed and re-affirmed procedures and ideas in these documents as important for noise assessment and analysis. One recent conference (15) was sponsored by the U.S. Air Force, EPA and the Federal Aviation Administration (FAA). At that conference many topics, particularly related to aircraft noise, were discussed and many new topics were added. However, there was also a reaffirmation of the information and procedures given in the early above mentioned documents.

The purpose of the guidelines for preparing ElSs was to serve their purpose for an interim period of time and then be updated. The US Army and the Air Force as well as a few other federal agencies have written their own procedures for handling noise but most are quite similar to those in the original article. The guidelines were, of course, a simplification, and were designed to interpret the effects of as many noise environments as possible. That they cannot handle all situations was recognized and partly compensated for by the addition of procedures for handling "Special Situations." And the recommendations in the guidelines should be the minimum that is done in preparing EISs.

The EPA in the Levels document, after a brief discussion of the literature, point out that the main reason that other physiological effects (other than hearing loss) are not addressed directly is because "—the noise levels required to produce significant non-auditory physiological effects are normally much higher than the levels required to protect the public health and welfare from adverse effects on hearing or interference with activities (32)."

Noise exposures above 75 dB in the guidelines are treated differently. A separate weighting function is recommended in addition to the one recommended for predicting the percent highly annoyed (% HA). This function for predicting hearing loss yields a value referred to as the PHL, population weighted hearing loss. Johnson states:

> "With respect to using PHL, the word "potential" should be emphasized. Measuring sound outside of residential structures does not necessarily reflect the noise exposure of the residents inside such dwellings. However, exposure to 75 Lydn (yearly Ldn) is considered to cause a serious impact for a variety of reasons and certainly noise induced hearing loss is one possible problem of such high exposures. For those who might be upset with calling PHL a population weighted hearing loss, use of "potential health loss" might be more satisfying. The

point is that regardless of how you want to interpret PHL, it provides a practical means for focusing on the problems of noise exposure above Lydn of 75dB (40)."

The PHL, as well as other measures recommended in the guidelines, have been criticized as being unrealistically conservative low since an Leq of 85 is allowed in the workplace. The EPA responds to the criticism by pointing out that the various measures proposed in the guidelines were meant to be conservative, that they are guidelines not limits, and that they were proposed without regard for economic factors. Nevertheless, there are some scientists in the area of Bioacoustics who object even to a suggestion that hearing loss could possibly be considered affected at a level as low as 75 dB. Some members of the committee that developed the guidelines were not in agreement with the recommendation to begin reporting PHL at such a low level. The main objection was, in addition to the fact that there is little or no evidence to support such a position, it is not reasonable to think that people who live in areas where the Ldn level is 75 dB are going to be exposed to that level all of the time, since they will be inside much of the time and because of the attenuation provided by work and dwelling structures the level will be reduced considerably.

This is not the place to debate the level to be set for hearing loss, however, the idea expressed by EPA in many of their documents that the level for hearing loss may also represent the beginnings of the onset of other adverse health effects is debatable. Nevertheless, there seems little doubt that more investigators agree with that position now than when the position was first offered. From the health effects literature there are very few instances until recently where investigators or teams of investigators are willing to say where health effects start or indeed if they occur at all. The EPA suggests that they may start around 75 dB. The studies most discussed to support their position seem to those proporting to show peripheral vascular constriction at levels around 70 dB. Burns (6) and Kryter (46) have criticized such studies in a rather compelling fashion and certainly demonstrated that the burden of proof is on those that say such changes are adversely related to health. Kryter states:

"—in our own laboratory we have been unable to date to obtain continued autonomic system responses of subjects exposed to noise in experiments aimed at replicating some of Jansen's studies. I believe, at the time of this writing, that the psychological set of the subjects and their concept as to what stimuli in the experimental situation they are supposed to respond to, or that some inadvertent conditions of the subjects to the noises in experiments of this sort, may explain these apparent discrepancies in experimental findings. For example, we found that the average heart rate and freedom of peripheral blood flow were as much, if not more, related to the experimental sequencing of the test sessions than to the presence of quiet and noise per se.

"In any event, I think it fair to say that Jansen's contentions of: (1) no habituation in stress responses to repeated noises at levels above 75 dB(A) or so, and (2) continued stress throughout the duration of an intense noise are resting on meager, if not contrary, experimental ground (46)."

Therefore, there seems only weak evidence to indicate that adverse health effects begin at levels as low as EPA suggests. Although a rather large percentage (37%) of the population will be highly annoyed at an Ldn value of 75 dB, this is not a direct argument for adverse health effects. (This too is in question see Paschier-Vermeer(58) or Table 1). This is the way the situation stands at the present time, and the important conclusion from this section is: the **EPA does not directly consider physiological effects because they seem to occur at higher levels than hearing loss (or better, protecting against hearing loss also protects against adverse**

health effects), and there is no quantifiable data basis that can be used for calculating a dose/response relationship.

EPA and More Recent Studies of Cardiovascular Effects of Noise

These studies are discussed separately because (1) little reference has been made to them in earlier sections, (2) they probably represent some of the most analytical thinking about the effects of noise in general, and about the cardiovascular system in particular, (3) they interpret the literature and address the problems of future research at a greater level of complexity than has been often done previously. The first studies to be discussed are the studies by Thompson. These related reports are "Epidemiology, Feasibility Study: Effects of Noise on the Cardiovascular System" and "An Annotated Bibliography: Literature: The Effects of Noise on the Cardiovascular System" (84,85). Thompson has attempted to evaluate the studies of the effects of noise on the cardiovascular system in terms of criteria and with a team of evaluators. Not only are teams of researchers with diverse skills needed to conduct research in this area but apparently they are also needed to evaluate research. This is a very commendable approach.

This approach by Thompson is more objective and to the point and quite detailed in the evaluation. As you would have guessed from reading the sections on the opinions of the experts that not any of the rated studies are very good. In fact most are very bad. Thompson summarizes the results of the evaluation process as follows:

> "The relatively poor quality of the identified papers is reflected in the individual component and overall ratings of the reviewers — The proportions of studies meeting more than fifty percent of the evaluative criteria were as follows: on the noise component, 6% of the English studies and 11% of the translated research; on the health outcome component, 33% of the English and 32% of the translated research; and on the epidemiologic methodology component, 42% of the English literature and 11% of the translated studies. When the lowest of the three component scores is taken as the overall validity score, no study reported in the English literature and only one in the translated literature was rated higher than '4' on the 0-9 scale.—These ratings indicate that the literature is less than fully informative for the task of judging the association between noise and cardiovascular effects (84)."

Often factors beyond the control of the investigators contributed to the low rating received for their study. A good example of this are the studies by Cohen and coworkers on the relationship between aircraft noise in the classroom and blood pressure readings on children. Thompson states: "Unfortunately, this well designed survey suffered from a major problem which frequently plagues longitudinal research. A relatively high proportion of the noisy-school children with high blood pressure were lost to attrition. Thus, the data from the longitudinal and more powerful design is of little value in judging a causal relationship between blood pressure and noise (84)."

At the time, these reports by Thompson (84,85) represented a milestone in noise research and a precedence was set for future evaluations of research in the area. She has continued this work to the present day with participation's at the national and the international levels (86,87,88,89). The work she and coworkers have done for the U.S. Air Force seems particularly valuable since they not only reviewed the literature since her previous EPA work but they also discussed the feasibility of conducting studies with various types of experimental designs. She has been a

restraining influence on those that would rush to condemn noise exposure before it has gotten a fair "scientific shake."

Another report "Noise, General Stress Responses, and Cardiovascular Disease Processes: Review and Reassessment of Hypothesized Relationships" (56) was published a year before the Thompson studies. It was a very important article (book) and should be read by anyone conducting or otherwise interested in research in the cardiovascular area (along with the AF studies mentioned above). The authors manage to convey the complexity of the area and to examine physiological responsiveness to stressful stimuli in considerable detail. They point out (1) a conceptual reorientation in approach to the problem is needed, (2) more quantitative models should be developed, (3) limitations of the logistic model used in cardiovascular research, (4) difficulties and problems of physiological measurement, and (5) the need to address the problem at the level of complexity it deserves. They also indicate that studies to date have been limited in the application of control procedures. In particular, modern statistical procedures are seldom used and some of the new methods seem quite powerful for assessing the effects of noise on the cardiovascular system. Furthermore, analytical procedures are available for measuring noise (dosimeters) and physiological responses (portable cardiovascular monitors).

The argument can be made very strongly for the use of dosimeters in noise effects research. Few, if any, epidemiological studies to date have adequately measured noise exposure (86). Most studies measure the effects of noise on some dependent variable (cardiovascular or otherwise) without knowing the level, spectrum, and statistical distribution of the noise under investigation. The measurement of the noise exposure during recreational hours and in the home is essential before one can differentiate individuals according to the amount of noise exposure in the workplace (or elsewhere for that matter). Two additional EPA publications "Five-Year Research Plan for Effects of Noise on Health (17)" and "Detailed Research Plan: Cardiovascular Effects of Noise (10)" were plans and not critiques, gave no references, and had as much administrative as scientific language. As a consequence, these studies are less useful and should be considered perhaps as "lists" of factors that should be considered in noise research.

Most EPA articles and sponsored reviews (even the analytical ones) quoted a lot of statistics about the high level of noise exposure in the population and point out the tremendous toll on human life taken by strokes and heart attacks. They used guilt by association and generally convey a "let's get on with it" urgency. Attention is given to the complexity of the problem, alternative approaches, etc., nevertheless, one, after reading EPA plans, gets the idea that they "really believe that it is just a matter of time until the dose-response relationship is established."

There is some evidence that noise research has benefited from the publication of these early studies. Thompson (86) points out in her ICBEN summary of research in the physiological area that : "Unlike earlier studies, investigators controlled for major confounding variables such as age, obesity and family history of hypertension. It soon emerged that the more rigorous studies which adjusted for confounders were less likely to find adverse effects." Nevertheless, it still seems true that in most research there has been little attempt to control for any but the most obvious potential confounding variables, to use power statistics to estimate the number of subjects needed in an experiment to obtain statistical significance, to exercise appropriate scientific caution, and to adequately assess the noise levels used in the experiments. Instead

there has been an "overuse" of the concept of "stress." It has been used in a global sense without adequate definition. Nevertheless, because of this stress it becomes so biologically plausible for noise to have an adverse effects that investigators use "borderline statistical significance" and "weak evidence" to describe experiments in which no statistically significant effects were found or where the effects were contradictory, etc. Furthermore, there is a willingness by some to describe the prevalence of the effects in communities (country) without being very clear that there really is such an effect (29).

In reference to an article by Kent et al (43), Passchier-Vermeer states:

> "Kent showed in an extensive study among US Air Force personnel that 2.3% of those with the lowest hearing threshold levels showed cardiovascular diseases (determined from the ECG) compared to 8.4% of those with the highest hearing threshold levels. This corresponds to a relative risk of 3.7 (58)."

We object to this quote because it is contrary to the major findings of the main body of the Kent et al report.. Kent et al in their original report stated:

> "The summation of our analytical review strongly suggests that there was not a significant difference between the top and bottom 10% hearing level groups in the selected cardiovascular descriptors. Comparison of the diagnosed sensorineural hearing loss group and the group with no such diagnosis also revealed negligible differences in the cardiovascular parameters examined (43)."

Contrary to Passchier-Vermeer, I could not find any way to manipulate the data given in the Kent et al article to obtain the percentage values (2.3% and 8.4%) claimed. The values I obtained were nowhere near to giving a relative risk of 3.7. This should not have been done in the first place since it ignores the statistics, findings and cautions present in the original article. And I, for one, object to using the data in this way which on the face of it suggests rather strongly that this article gives evidence for an adverse effect of noise on cardiovascular function, when, in fact, it does the opposite.

Physiological Response Change as an Index of Adverse Effects of Noise

It is very difficult to generalize from short-term studies using human subjects in which they are exposed to noise and effects observed in their physiological response system. The jump from these studies to judgments or predictions about what will occur in the "real world" is a long one. There is a real problem in interpreting the meaning of a short-term physiological change. Furthermore, it was stated a number of years ago at an international symposium on the health hazards of noise that no more studies are needed (to determine the effects of noise) in which only a single physiological function is measured (38,39). It was argued that such studies do not contribute to our understanding. This is a direct recognition of the fact that the human physiological response system is so inextricably interrelated that it is naive not to consider this interrelationship. There are such complex feedback networks within the human physiological system (particularly, the cardiovascular system) that it is quite difficult to determine a pattern of physiological response to even a relative simple stimulus (47). This point is worth further

consideration and discussion because in much of the noise literature, the complexity of the physiological system is not recognized. The complexity can be demonstrated by the conclusions of Weybrew in his review of "Psychophysiological Response to Military Stress (96)." In his review, he considered such potent stresses, among others, as heat, noise, vibration, weightlessness, acceleration, hypo-hyperbaric atmospheres, hunger and thirst, radiation, and threat to "life and limb." He states:

> "It may be tentatively concluded from this review of field studies of this type: (1) In general, psychophysiological changes do not correlate significantly with performance decrements, (2) Psychophysiological changes do not occur in all persons in a given stress situation, (3) If changes occur, often they are not in the same direction for all subjects at a given time, and (4) in some instances, the direction of changes may not be the same for a given subject measured over a period of time. Concepts proposed to explain these wide ranges of individual differences in response to stress vary a great deal, however, there are some workers who apparently feel that most of the differences in response to operational stresses of this kind result from differences in the subjects' knowledge and expectation regarding the duration of the task or mission as the case may be (96)."

Additional articles contained in the same book as the above-referenced article demonstrate the poor correlation obtained between different physiological responses and the role that cognitive appraisal and coping mechanisms can play in changing physiological responsiveness (48) to the same situation. Further, it was demonstrated that the acceleration of the pulse rate was opposite in direction depending on the task the individual was engaged in when the stimulus was presented (47).

It is becoming common knowledge that the anticipation of an event, both subjectively and physiologically, can be worse than the occurrence of the event itself (i.e., a trip to the dentist). Similarly, the anticipation of an increase in noise level can for some people be more stressful than the actual increase in noise. The manner in which a person reacts to increased environmental stimuli results not only from the increase in stimulation but also by the type of biopsychosocial individual that is doing the reacting. Each person has his own life style, his own highly individualized beliefs, coping mechanisms, attitudes and physiological and biochemical individuality. What the person **"is"** may be more important in the reaction to aversive stimuli than the increase in stimulation. Therefore, one can see the difficulty in generalizing from such studies of short-term physiological change to actual situations in the real world. The time is past for such simple-minded reasoning that is exemplified by most lay discussions of noise effects and by discussions on the health effect of noise where they point out all of the physiological systems adversely affected (at least by implication) by noise. What they don't make clear is that these same physiological changes can occur as the result of exercise, visual stimuli, positive events, etc. A demonstration that noise produces physiological changes is not a demonstration that noise has an adverse effect on health. Most people know this but many still write and speak as if this is the only information necessary. This type of thinking stems from the outmoded concept of sensory overload and is augmented by the biological plausibility of such a supposed noise effect. The idea being that one is so overwhelmingly stimulated with sensory bombardment that the physiological system is overloaded and over responds. It is difficult to think of many situations where the physiological system of man is over stimulated by noise.

It was pointed out above that it was recommended that we are not in need of studies that use only one physiological measurement. Nevertheless, for ease of discussion, consider just the measurement of blood pressure (the same tenor of the discussion could be applied to almost any

physiological measure). It has been pointed out that blood pressure would be a good physiological measure to use in noise research because it is so simple to measure. Contrast this statement with one made after a brief discussion of the problems in determining a valid and reliable measure:

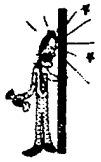
> "---Unfortunately, many measures are so variable over time and occasion that change may be difficult to see. For example, blood pressure, particularly systolic pressure, can differ markedly because of anxiety level, time of day, body posture, and many other factors. In fact, blood pressure is so variable that a diagnosis of hypertension usually cannot be made on the basis of a single reading (73)."

The author also goes on to point out that blood pressure is such a imprecise measurement that with repeated measurements that a clinician's idea about what the blood pressure ought to be can influence the actual reading of measurements. The next time you take your own blood pressure at Wal-Mart, notice the written warning about the variability of blood pressure. It can vary from 10 to 30 ml of mercury in a matter of 10 minutes. Thompson et al point out:

> "Among the more common findings of cross-sectional and ecologic studies— are slight elevations in systolic (SBP) or diastolic blood pressure (DBP). Mean increases as great as 6 mm Hg in DBP and 9 mm Hg in SBP, and unadjusted prevalence ratios of 1.5 to 1.7 for high noise relative to low noise exposure, have been reported in such studies. Considering the DBP in the general population of healthy adults has a log normal distribution with a mean of about 80 mm Hg and a standard deviation of about 15 mm Hg, and the SBP is similarly distributed with a mean of about 120 mm Hg and a standard deviation on the order of 20 mm Hg, these changes of less than half a standard deviation cannot be considered very alarming, particularly when it is unknown whether they are transient or persistent changes (87)."

We also do not know how the changes are related to changes caused by common stimuli such as exercise, laughing, etc., but generally the changes to noise as indicated above have been small. Many experimenters in the past have made little or no attempt to control for factors that could confound blood pressure measurement. Schwartze and Thompson (72) point out that because of the improved technology, it is possible to get better and more accurate physiological measures than in the past and although; this is true, I have not seen much indication , with a few exceptions, that investigators have used these procedures. In many experiments there has been little or no attempt to control for potential confounding variables.

Noise as a Stressor and the Study of Noise in Relationship to other Stresses



Earlier, the idea was put forth that we should not be overly concerned with the immediate vegetative responses of the body to noise, the primitive autonomic response, i.e., the classical "emergency response" of Cannon. Research results seem to indicate that it is rare that one would ever expect to obtain a primitive response to noise; even physiological responses at a primitive (autonomic) level are conditioned by the individuals' orientation (set) and appraisal processes (8). This point has been emphasized previously and is also discussed in a review by Kryter (46). It was known more than 35 years ago that the most intense physiological responses to stress often occurred "before" the actual experience. This has been observed in military and

sport parachutists, in the Project Mercury Astronauts, and in the high altitude balloon flights to the edge of space. If one would suggest that the immediate, vegetative response to noise is intense then one should examine the levels observed under the above-mentioned conditions. For example, Trumbull, in a discussion following the presentation of a paper on Adaptive Stress Behavior (69), and concerning the high altitude balloon flights states:

> "I would like to make an observation about something Dr. Ruff was saying concerning anticipation. We had an experience in our high altitude balloon studies where we were doing some biotelemetry. Pulse rate and blood pressure were both being recorded in a plane that was to accompany the men in the balloon. A decision had been made that the balloon would be ripped and brought back to earth by remote control from the plane when these physiological measures got above what the medical men felt was a feasible level for them, both in terms of rate and pressure. While the balloon was being filled on the ground, with Ross and Prather in the gondola, both the pulse rate and blood pressure indices rose suddenly far beyond the level that had been set for aborting the entire flight! They were asked what was going on—had they checked their instruments, was something wrong, were their contacts in good position, etc. They checked them and all was in order. They were just preparing for the flight. Apparently, merely in anticipation they had already exceeded the critical levels (which in fact they never did again during the entire flight) (90)."

Similarly, Ruff and Korchin (69) have suggested that the high physiological arousal of the Project Mercury Astronauts was their way of getting ready for the space flight and they further pointed out that the astronauts actively sought out situations that produced such high arousal (i.e., in training simulators and in flying jet aircraft). And it is well known that some people actively seek out and enjoy highly arousing situations that are considered too risky by other people.

Investigators, in most areas of stress research, guard against considering physiological activation (or change) as bad without considering the psychological and social context in which such changes occur. For example, F. Cohen in discussing reviews of the literature states:

> "Most of the literature relating psychological factors to disease emphasizes the negative effects of increased physiological activation. However, there may be positive consequences as well, and the interplay of these positive and negative factors needs further investigation. For example, Frankenhaeuser (1975, 1976) reviews studies that show that those who habitually secrete high levels of epinephrine have higher IQ and better school performance, are rated as happier and livelier, score higher on tests of ego strength, and perform better on certain laboratory tasks, as compared to those with low epinephrine levels. Frankenhaeuser (1976) and Gall and Lazarus (1975) also suggest that the magnitude of the physiological response may be an inappropriate measure of adaptation compared to the time necessary for the return to base-line hormonal levels. Thus, good adjustment may involve both efficient mobilization and demobilization of physiological systems (9)."

In the last 40 years, research like that quoted above has increased so much that it would be impossible for one individual to read all of it even if he spent all of his time on it. And from the popular press we have been bombarded with articles on stress. Sarason states: "There are few topics more "in" than stress. It sometimes seems next to impossible to avoid what seems to be continual bombardment of suggestions and prescriptions for avoiding or coping with stress." He goes on to point out that he likes the "Handbook of Stress" because it doesn't attempt to give easy answers and states: "One of the unmistakable conclusions to which the Handbook of Stress leads the reader is that stress almost always must be defined in multidimensional terms." Later in the review, he states: "Thus, there are intertwined physical, psychological, and social pathways to illness vulnerability.—The section devoted to Basic Biological Processes deals as much with psychological as with biological processes (71)."

Some pioneering work has been conducted in this area by Glass and Singer (18) and followed by the work conducted by Cohen and coworkers on the effects of aircraft noise on the blood pressure of children (see Thompson for a summary and critique of the Cohen et al studies).

These articles have not been ignored and the results have been often quoted in reviews and by the popular press. In the popular press they have, on occasion, been exaggerated to the point of producing a "scare" article. The results of the studies should not be used to scare anyone since the generality of the findings has not been determined (see Loeb (50) and Harris (26)). Furthermore, it is doubtful that more publicity of these findings would be fruitful without more data. These articles do represent an innovative approach to the study of the adverse effects of stress, and of noise in particular. In more recent years, this is an even more lively topic but with few clear-cut effects. Nevertheless, there is been a greater willingness to discuss the multidimensional nature of stress stimuli. Babisch et al point out in their study of traffic noise that:

> "In accordance with the stress model, variables of disturbance and annoyance may be better predictors for cardiovascular effects than the noise levels. Figure 2 which refers to the Speedwell study supports this idea. In addition to the association between traffic noise level and IHD incidence, questionnaire item sum scores of annoyance and disturbances due to traffic noise are incorporated into the graph. In the highest category we find a steep increase in relative risk in these subjective determinants of exposure. Our future analyses of the Caerphilly and Speedwell studies will continue to focus on this. However, from the decision making point of view we have to bear in mind that annoyance cannot be regulated, but noise level can. Therefore every noise research should in some way relate to the noise level. (5)"

a. Performance and Physiological Studies of Impulsive Acoustic Stimuli

One of the most often made statements by people wishing to emphasize the hazardous effects of noise is that noise initiates immediate reactions of the autonomic nervous system and that these "vegetative" changes are hazardous to the health of people. As seen in the expert opinion section of the present discussion, Kryter has pointed out "There is no likely damage risk to a person from the possible unconditioned stress responses to noise that are mediated by the autonomic nervous system (46)." He reaches this conclusion because of the rather small changes in physiological indices that have been demonstrated by brief exposure to noise. For example, in describing one experiment where subjects were exposed repeatedly to 5 sec pulses of white noise and of a 1000 Hz tone at 85, he states:

> "—the magnitudes of the physiological changes that are associated with these responses are rather small in comparison to the range of physiological responses or states observed in human organism during homeostatic operations of the autonomic system normal to daily living. For example, in regard to this point, the greatest heart rate change — is about 11 beats/min, from 75 to 86, and this for only 1 or 2 beats, and the peripheral blood volume changes last but for 10-20 sec or so. Consider that changes much greater than these occur from mild exercise, fright, sudden changes in air temperature, laughter, etc. (46)."

Burns (6) reaches a similar conclusion as Kryter and both point out that the often quoted studies on peripheral blood flow by Jansen are not consistent and that they do not support an interpretation of an adverse effect on human health (also see Thompson (85)).

Most of the studies reviewed above have used impulsive stimuli that are quite different from the pattern of acoustic energy produced by sonic booms Furthermore, most studies did not elicit a startle response as sometimes occurs to a sonic boom. A series of studies by Thackray and coworkers (80) are important, in this regard, because they have exposed people to sonic boom both in the field and in a laboratory simulator. Thackray et al (80) found in one study that simulated sonic booms as experienced indoors and having outside overpressures of approximately 50, 100, and 200 N/m2 (1.05, 2.1 and 4.2 psf) had no adverse effects on tracking performance. "On the contrary, the booms appeared to increase task attention and facilitate performance." The increased attention overcame a short temporary disruption. Subsequently, they used a more analytic measure, a "steadiness test" and reached the tentative conclusions "that sonic booms experienced indoors may cause slight arm-hand startle responses which could have adverse effects on occupational tasks in which arm-hand steadiness is the principal skill involved, but that it seems unlikely that these responses would significantly impair performance on less sensitive psychomotor tasks (80)." The amplitude of the arm-hand movements produced by startle yielded a mean value of only half a centimeter. These authors do not discuss the probability of an accident that could result from the startle movement, but considering the small amplitude of movement it would seem unlikely that sonic booms would produce many accidents; nevertheless, much more research is needed before that problem can be adequately addressed.

This study is also of interest for comparison with the results discussed above by Burns and Kryter, since Thackray and coworkers (80) measured pulse rate in their experiment, but first a discussion of the noise stimuli used in the experiment. They state "two levels of outside overpressure were studied (50 and 150 N/m2) (1.05 and 3.15 psf) with rise time held at approximately 5 msecs for each level. These overpressure levels approximate the range of overpressures (75 to 175 N/m2) expected along the centerline of the sonic boom carpet for the Concorde SST and the rise time was virtually the same as the median rise time (6 msecs) reported for the XB-70 during the Edward's Air Force Base test." The indoor noise measurements for the two levels of boom were 105 and 111 dB respectively. They report that these levels were obtained with a Bruel and Kjaer Type 2204 Impulse Sound Level Meter set on Impulse Hold Position. One would think that this would approximate a C level weighting, since the dBA levels were reported to be 74 and 83 dBA. Separate groups of 10 subjects each were exposed to 6 booms at each level of boom. The subjects ranged in age from 18 to 29 years. These authors report "Magnitude of heart-rate change was determined by taking the difference between the maximum heart rate in the five-second pre-stimulus and post-stimulus intervals. They found a decrease in heart rate for the 50 N/m2 condition of -2.43 beats per minute (bpm) and an average increase of 3.4 bpm for the 150 N/m2 condition. Subsequently, both groups of subjects were exposed to a pistol shot. The first group showed a change of 10.9 bpm and the second group showed a change of 11.6.

These authors describe further research as follows:

> "It is interesting that similar results for the arm-hand response were also obtained to actual sonic booms experienced indoors during a recent field study of startle effects. Flyovers of supersonic aircraft generated booms having outside overpressures ranging from 60 to 640 N/m2 (1.26 to 13.44 psf) with a mean rise time of 2.5 msecs were examined. Mean arm-hand response amplitude (using the same task used in the present study) was virtually the same in both studies. Although there was evidence in the field study which suggested a slightly greater amplitude of response to the more intense booms, the primary effects of increases in overpressure was an increase in the percentage of subjects (Ss) showing startle reactions. Percentages of subjects exhibiting startle reactions to the 50 and 150 N/ms booms in the present study were almost identical to the percentage obtained for comparable overpressures in the field study (12% and 62% respectively) (80)."

Taken together, the results of both studies suggest that the indoor stimulus intensities, for the range of boom exposure levels employed, were or only slightly above the threshold for evoking startle reactions strong enough to involve arm-hand responses. Higher overpressure levels simply evoked these marginal startle reactions in a greater number of Ss than did lower levels, with little or no evidence of a corresponding increase in response amplitude. The startle reactions are considered marginal since in both the present study and the field study, arm-hand response to a "standard startle stimulus (.22 caliber pistol shot) was approximately twice the mean amplitude obtained to the booms (80)."

The above results seem to agree with those by Lukas and Kryter (51) who found no significant impairment in tracking performance when subjects were exposed to booms, and with those of Rylander and coworkers (90) who found very little effects of booms on automobile-driving performance in a simulator.

b. Early Exploratory Studies of the Effects of Sonic Boom

Some people in public meetings about boom exposure often suggest that they are "guinea pigs." That nothing is known about the effects of sonic booms on people and they are being exposed to booms to find out what the effects really are. Actually, quite a bit is known about predicting booms and about possible effects of booms. The data are not orderly, but over 20 years ago von Gierke and Nixon (91) concluded "The probability of immediate direct physiological injury to persons exposed to sonic booms in the community is essentially zero. Long-term effects on health of repeated daily exposures to sonic booms have not been investigated." And since then little data has been obtained on the question. However, it would be tempting to conclude that there is less reasons to expect health effects from sonic boom than there is from other types of acoustic stimulation. Unfortunately, no unequivocal data is available on any type of noise at levels in the range we might expect a community to be exposed, and there is still the question of the unexpectedness of the stimulus (resulting in the startle response) and the possible indirect effect of loss of sleep. Nevertheless, some early studies would suggest that sonic boom, even at

very high intensities, is not the threat that many would interpret it to be. In one exploratory study conducted by Nixon and coworkers (54), the primary author and his associates as well as various other people were exposed to sonic booms ranging in peak positive pressure from 50 psf to 144 psf. The subjects ranged in age from 6 to over 70 years. In some cases, the F-4C aircraft passed less than 100 feet directly above their heads. These experiences were described as follows:

> "The pressure waves were felt by the entire head and body during boom exposure as a jarring sensation. Rather strong tactile and kinesthetic stimulation were experienced as well. Some momentary discomfort, fullness, and ringing of the ears were experienced with the more intense booms and these persisted from a period of a few seconds to as many as 60 to 120 seconds. For the most intense booms the symptoms of fullness, ringing, etc., were significantly greater in the ear facing the approaching aircraft than in the contralateral ear. Symptoms were essentially the same for both ears for the lesser intense booms.
>
> "No distinct auditory pain was reported although some booms were described as very sharp. Personnel further commented that the most intense booms would have been judged to be painful had they been any greater in magnitude. From this the threshold of pain for these individuals and kinds of exposures were perhaps close to but still greater than 144 psf. Although hearing acuity was not physically measured, subjects reported no indication of any observable symptoms of temporary hearing loss or other ear involvement.
>
> " Individuals performing routine tasks of photography and operation of the electronic equipment were required to visually follow the aircraft during its supersonic pass. Although task performance was not interrupted or bothered all personnel expressed avoidance behavior consisting of involuntary ducking and flinching in response to the boom experience.—Startle responses to the actual pressure wave also occurred. This behavior did not habituate during the three-day flight program. In fact involuntary tensing or muscle set of the body in anticipation of the booms appeared to be stronger for the later exposures than during the initial boom experiences (54)."

The booms that these individuals were exposed to were many orders of magnitude higher than would be expected to occur in the community, even the lowest level! Therefore, if any should be classified as guinea pigs, it would have to be the above individuals who participated in the study, and they all volunteered. Furthermore, even though this study was an exploratory one and concerned only with subjective impressions, it does help set in perspective the effects of the levels of sonic booms that are expected in the community.

Another study (unpublished) conducted by the Civil Aeromedical Research Institute in 1964 studied the effects of sonic boom on hearing. They report exposure of 23 healthy male subjects, ages 26 to 60, to approximately 600 sonic booms of up to 16 psf intensity (a few were accidentally of higher intensity) between November 18 and December 15, 1964, produced no detectable alteration in hearing acuity at 500, 1000, 2000, 3000, 4000, and 6000 cps as measured with a Maico audiometer."

Both of the above studies were exploratory in nature and are most notable for the very high level of booms that individuals have been exposed to, without apparent harmful consequences.

Numerous other studies have been conducted, the St Louis study, the Oklahoma City study, and others have evaluated the response of people to sonic booms (summarized and evaluated in von Gierke and Nixon (91)). One study has been conducted in which the investigators were interested in assessing the effects of sonic boom on the health of people. This study (2,41) was conducted in Nevada because people there have been exposed to more sonic booms than any other place in the United States. Estimates of the levels of sonic booms (in yearly DNLs) were obtained for the years 1969 to 1983. The highest levels at any location only yielded DNLs of 50

to 56 dB. These levels were correlated with the available epidemiologic data which unfortunately was not only incomplete but consisted of only morbidity and mortality data. Under these circumstances, it is not surprising that no adverse health effects could be attributed to sonic boom. The authors conclude:

> "In summary, this study has clearly demonstrated the viability of acquiring and analyzing the global measures of sonic boom environment and health effects utilized for this study. However, it has also demonstrated that the specific global measures employed in this study do not show any evidence for the existence of possible health effects due to sonic boom exposure. Any such evidence, if it exists, is most likely to be found only in a prospective study monitoring a substantial sample of individuals over a sustained time period (41)."

c. Effects of Noise and Sonic Boom on Sleep.

Interruption of sleep is a very important problem. Many people are quite concerned about their sleep as witnessed by the large volume of prescription and over the counter drugs sold either to help people go to sleep or to enable more restful sleep (or both). Little is known about the effects of sleep loss on human performance and although, not much is known about how sleep loss affects health, there is little doubt that acute loss of sleep can adversely affect health. In spite of quite a few laboratory studies of the effects of noise (and even a few using simulated booms) on various aspects of sleep, there have been few field or epidemiological studies of sleep. von Gierke and Nixon state:

> "Sleep interference from nighttime booms, which may be a major determinant of public reaction, was observed for simulated sonic booms in excess of 1.0 psf for which adaptation did not occur during the relatively short test period. All sonic booms in that study were adequate stimuli for awakening subjects during their REM (Rapid Eye Movement) state of sleep. Sleep interference by booms is to some extent dependent on the individual. Subjects about 70 years of age are more likely to be awakened by simulated booms than are younger subjects around 47 and around 8 years of age. Possible long-term effects on sleep of repeated nightly exposure to sonic booms are not known (91)."

It is possible that older people are more likely to be awakened by booms (and other types of noises for that matter) and even to stay awake longer after sleep interruption than younger people. This seems a logical idea and some data seems to lend some support. However, as in most areas of noise effects research, the evidence is not unequivocal and much more research needs to be done before we can predict levels of booms or other types of noise that are likely to interfere with sleep. Goldstein and Lukas state:

> "There is little argument that noise may disturb sleep. However, such a broad qualitative description is of little use in assessing either the magnitude of a noise impact or the benefits expected from lessening environmental noise. Indeed, for noise control purposes, it would be more helpful to have quantitative measures of the extent to which noise may affect a person's sleep. These measures should be quantitatively (and statistically) documented criteria or cause-effect relationships. Using such criteria, the probability, magnitude, or incidence of a noise-related sleep disruption could be predicted from a knowledge of the noise exposure (19)."

Their recommendation, that we need to "identify particular subgroups of the population more likely to be affected by noise during sleep (such as the elderly, ill, day sleepers, etc.)," still seems particularly important.

The Day-Night Average Sound Level (DNL) seems to be the proper methodology for assessing the noise impact for the vast majority of Air Force aircraft operational scenarios as well as around civilian airports.. The 10 dB night-time penalty levied against noise during 2200-0700 in this metric is designed to specifically account for the intrusiveness of noise during this period and the potential impact on sleep. However, an unusual number of night-time noise events may warrant that supplemental information be included in an environmental assessment. For such cases, until substantially more research has been conducted, an equation developed by Harris and Speakman based on the data gathered by Pearsons et al(61) had been adopted both for use in Air Force Environmental Assessments (EAs) and Environmental Impact Statements (EISs) and by FICON for determining the potential impact of aircraft noise on sleep interference. Pearsons et. al. (1989) in their report "Analysis of the Predictability of Noise-Induced Sleep Disturbance" summarize the results of 21 published studies on sleep. They state: "—a lack of appropriate field studies, combined with large discrepancies between laboratory and field studies, precluded development of such a model." Certainly, there can be no question that percent awakened for laboratory studies was vastly different from that for field studies. Separate linear fits to field and laboratory data points also give greatly different predicted values from their regression equation. Moreover, neither equation seems to give high enough awakenings for relative large indoor Sound Exposure Levels. For example, an indoor SEL of 90 dBA gave percent awakenings of 5% for field and 42% for laboratory studies (see Figure 1. below).

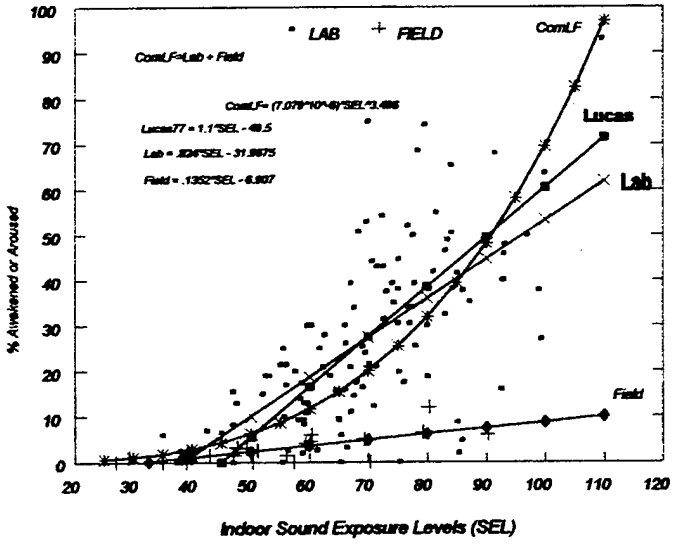


Figure 1. Lab and Field Curves of Pearsons et al (61) along with combined Lab and Field derived curve, and early curve used by Lucas.

Rather than accept a one number criterion (indoor SEL= 45 dBA) as has been proposed, an attempt was made to obtain a better fit by grouping the data. With variable and incomplete data bases the values are sometimes grouped to reduce variability. In the present case, the data were grouped not only because of the variability but also because values were under represented at the higher SEL values. By grouping the data by 5 dB intervals across the range of approximately 30 to 110 SEL, each class interval can be equally represented in the range. There were seventeen intervals, but two had 0 cases, therefore, the regression was conducted on the middle of the 15 remaining SEL class intervals versus the mean % awakened in the particular interval. This procedure give equal weight to each of the class intervals regardless of the number in the particular intervals. If data values are evenly distributed across a range then this grouping by class interval produces results that are quite similar to what one would have gotten if the individual values had been used. This, of course, was not the case for this data base of 136 values since, we knew of the under representation at the high SELs. The best fit to the data, grouped in the way described above, was given by a power function. The equation is % Awakenings = .000007079 x SEL ^ 3.496 (see Figure 2. below).

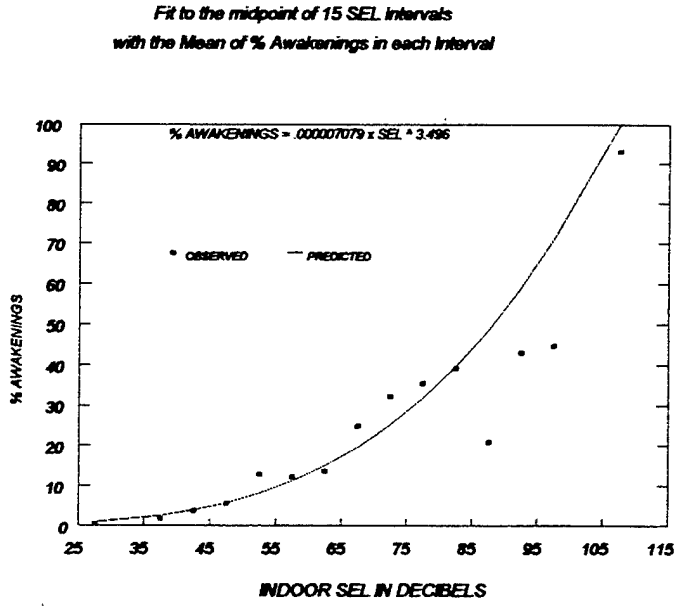


Figure 2. Fit to the midpoint of indoor SEL interval values and means of the %awakenings in the interval (taken from Pearsons et al(61)).

Although this curve is used at the present time, it may be subject to replacement in the near future. In a recent study in the United Kingdom of aircraft disturbances of sleep, Ollerhead et al (57) obtained results that seem to fit in well with the field research surveyed by Pearsons et al (61). They used a very promising measure of sleep disturbance or behavioral awakening that could result in a more economical way to conduct sleep research in the future. . Nevertheless,

many questions remain about what they are actually measuring and how it relates to measures of behavioral arousal from sleep that have been conducted in the past.

The proposed curve above is more conservative and seems to predict a more reasonable number of people awakened at each SEL than does the field curve of Pearsons et al. However, the curve probably has more intuitive appeal than it does scientific support. And we would certainly agree with Pearsons (61) that: "One disconcerting factor remains about sleep disturbances. We still don't know how awakenings or sleep stage changes relate to long-term health effects. It may be that the annoyance created by sleep disturbance is the most appropriate measure after all."

In spite of only a preliminary (at best) understanding of a dose-response relationship between noise and behavioral awakening, sleep interference is one of the four factors that we can use to evaluate the severity of noise environments (the other three are annoyance, noise-induced hearing loss, and speech communication) (93).

The effects of noise on sleep has been a long studied area of noise research and in the future more can be said about the effects of noise on sensitive groups, on after-effects, on interactions with other variables, on going to sleep as well as on awakening, and effects during the most sensitive time of night.

d. Military Training Route Noise (High Speed Low Altitude)

Noise along Military Training Routes (MTRs) because of the low flying aircraft differs in a number of ways from conventional noise around airbases. These differences include the number of daily flights, their occurrence in time, their onset, duration and decay times, and their intensities and spectral characteristics. The most important of these factors seems to be the onset rate of the flyover. In preliminary observations, high onset rates seemed to create startle or "surprise" and contribute directly to the observers perceived annoyance (63,64,65). As a result of these observations, an interim noise metric was recommended for evaluating the potential annoyance of communities to MTR noise environments. This onset rate adjustment provides a noise penalty to account for increased intrusiveness because of the surprise factor of low level, high speed aircraft operations. The metric is based on the best available evidence, however, research is continuing (25,79) to better account for the above factors. These studies were and are continued to better predict how people would react to MTR noise in terms of their annoyance. We were quite surprised by European research (33,34,35,36,74) that, along with a few other factors, claimed to have demonstrated that the noise from low flying aircraft adversely affected the hearing as well as the cardiovascular health (blood pressure) of people.

We will consider these factors, one at a time. The reason that we were not concerned about permanent hearing loss from low flying aircraft was first, studies of hearing loss produced by

sonic boom (see above) indicated no permanent loss and although, the noise from low flying aircraft is not technically impulsive noise, at the higher onset rates it is somewhere between stationary and impulsive noise (58). However, we do question statements about onset rates of 180 to 200 dB/sec occasionally being found and believe these occurred because different segments of the same rise time signal were measured. This is quite different from typical procedures for measuring rise time. Second, in the process of measuring flyover noise we have been exposed to levels higher than those suggested in the criteria with no subjective impressions of hearing loss, ringing or fullness in the ears. Certainly, these are anecdotal remarks and objective audiograms were not taken, however, we did experience levels, where the aircraft flew over at 100 feet and produced levels as high as 128 dBA noise intensity. We have experienced several of these flyovers on the same day with no obvious bad effects.. We find a criterion of one flight per day at 115 dB to be needlessly conservative (34,75).

A recent study in our laboratory where TTS was measured seems to support our subjective impressions of little or no TTS at some fairly high simulated flyover noise levels (55). In this study "—Worst case flyover noise exposures that exceed those that might be experienced in a real world situation under a low level military training route were utilized as stimuli in the study." The noise stimulus was a recording made 108 feet under an F4 aircraft flying at 579 knots at a MTR in Europe. The stimulus was presented at noise levels from 115 to 130 dB(A). After a familiarization period (phase 1), subjects in the second phase were exposed to 8 flyovers of 125 db(A) in session one and 8 flyovers at 130 dB(A) in session 2. In both sessions the eight repetitions were separated by about 90 seconds. Hearing threshold were measured pre exposure and 2 minutes and 1 hour post exposure. The authors conclude: "—Significant changes in hearing levels due to these exposures were not observed ." In Recent research begun by Johnson (59) which shows less TTS at some very high impulsive noise levels than one would have expected from the TTS literature also supports our position as well as existing criteria used to protect against hearing loss. Third, the noise criteria we have available suggest that flyover noise levels are not endangering the hearing of people. In the publication "Combating noise in the 90s", it is pointed out that: "The recently adopted international standard, ISO: 1999, provides information on the efficacy of this occupational noise regulation. Evaluation of the risk of sustaining a hearing handicap for individuals exposed chronically below 90 dBA suggests that the occupational noise standard, as it presently exists, does provide protection against NIHL (9)." These authors argue that no studies are really applicable to the hearing loss produced by jet aircraft in Germany. "The only remaining approach, then, is a comprehensive search of the literature regarding damage to hearing, with an attempt to build upon this in order to recommend a hearing damage risk criterion with regard to low-altitude flight noise."

The authors reject out of hand those studies that contain measures and data, on which have been based standards (ISO 1999) for evaluating hearing loss. These studies and standards seem most relevant to what they are trying to predict. We don't believe that there is a great difficulty in applying ISO 1999 to low altitude jet overflight noise. The authors, on the other hand, state that these studies are not relevant based on a review of studies based primarily on animal data. Furthermore, some of the conclusions based on those studies are not firmly established, would be rejected by a large part of the acoustic community, and are tentative at best. Particularly, the studies on the combination of other types of noise with impulsive noise in producing hearing

loss. This is certainly not a straightforward approach that these authors have taken and one that we don't believe is supported scientifically. They point out that there are three basic types of noise; (1) Slowly changing noise; (2) Greatly fluctuation noise - some where between continuous and impulsive noise; and (3) Impulsive sound-such as gunfire or a pile driver. They argue since there is no one way for assessing the hearing loss for all three types of noise, an alternate approach must be taken. This does not make much sense because the ISO Standard 1999 clearly states that it is possible to extend the standard to all types of noise. This standard was written and has contributions from many of the world's foremost experts on hearing loss. These authors reject this standard without good arguments. Similarly, we know of no evidence that makes people more sensitive to hearing loss because they live in areas with low background noise levels. Furthermore, the statement—"the noise from jet aircraft almost always occurs unpredictably, neither the natural auditory protective mechanism (contraction of the muscles of the middle ear triggered by fluctuating noise of intermediate intensity and resulting in an increase in impedance) nor active responses (covering the ears) becomes effective in time. The noise from low-flying, high-speed jet aircraft—assuming a low background level—almost always hits readapted ears at their full sensitivity, suggesting its characterization as a more impulse-like noise." This neglects the fact that most jet flyover noise will be heard while people are inside structures (home, workplace, shopping center, etc.) where the noise from the flyovers would be considerably attenuated as well as having a much higher background noise level. Similarly, many Audiologists, with backup data, would question the assumption of no acoustic reflex to jet flyover noise. Nevertheless, if one should accept that jet flyovers sometime appear like impulse noise, even though the rise times do not approach those of gunfire, sonic boom, and most industrial impulse noise, then ISO 1999 gives ways of evaluating the effects of such noise. Why do we need another way for evaluating this noise based on questionable assumptions.

In Figure 3 below , the eight-hour Leq is plotted against the log number of movements for Single Event Levels (SEL) of 115, 110, 105, and 100 dB. The actual number of movements are above each data point. Twenty-eight movements at a SEL of 115 dB gives an $L_{eq,8h}$ of 85 dB while 911 movements are necessary at a SEL of 100 dB to reach an $L_{eq,8h}$ of 85 dB. The 85 dB $L_{eq,8h}$ level is the level where less than a 10 dB Noise Induced Permanent Threshold Shift (NIPTS) would be produced in the top 10% of the population for the frequencies of 0.5, 1, 2, 3, 4, and 6 kHz after a 40-year exposure according to ISO Standard 1999. Not included in the figure but it can easily be calculated to show that 9 flights with a SEL of 120 dB are required to reach an $L_{eq,8h}$ of 85 dB. Similarly, 3 flights a day at a SEL of 125 dB would reach an $L_{eq,8h}$ of 85 dB. It is important to recognize that these values are for levels directly impinging on the human ear. If one were inside structures for some of the overflights then these flights would be attenuated by 10 to 30 dB depending on the structure. It is also important to realize that according to ISO 1999, these are the number of flights that can occur for at least 5 days a week (the work week), every week for 40 years.

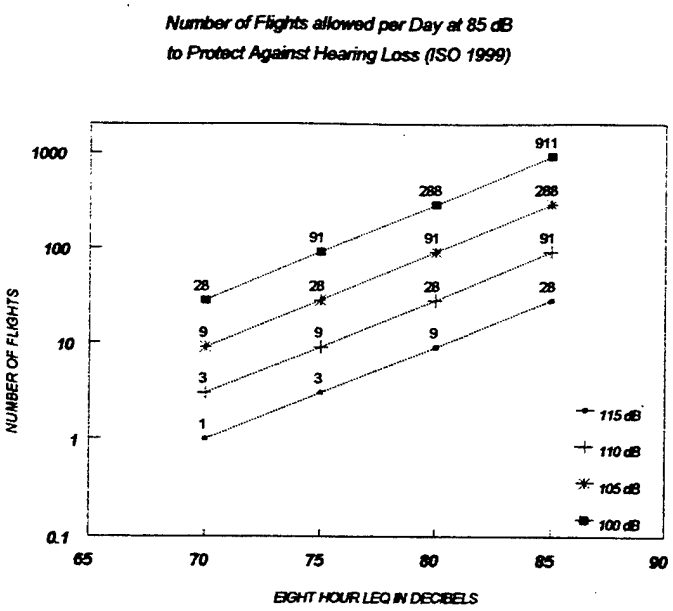


Figure 3. Number of flights allowed per day at SELs of 100 - 115 db for 8 hr LEQs of 70 - 85 dB.

We cannot respond to every issue discussed in the present research but we can say it is based on questionable measurement techniques such as having people in various sections of the country give an estimate of the number of flights they heard in a given time period where the time periods were not consistent group to group and linear extrapolations were used in some cases to give the number of movements in an 8-hr day. Then reports are given for half a day after the 8 hr extrapolation. There is a most confusing presentation of graphs, tables, and discussion. One is not really sure what data they are discussing; actual measured noise data, estimated data, or theoretical data. And we have a hard time agreeing with the suggestion of these authors that broad band noise may cause more hearing loss in the long run than narrower band and pure tone type of noise. Their statements are laden with the appropriate qualifiers as they should be; however, the very fact that these qualifiers such as "may possibly result" and "more or less true" seem to have had no effect on their recommendations, since they have proposed a criterion that is so restrictive in terms of protecting hearing loss that we can not imagine that most audiologists and others expert in the study of hearing loss could accept it. These authors provide little discussion of factors that could alter the adverse impact of the noise on human hearing. First, there is still a great deal of debate on the value of animal research on hearing loss in generalizing to human hearing loss. The authors spend a great deal of space telling why animal studies are appropriate for the study of human hearing, however, they do not mention the many and well known cautions in the literature against making such generalizations. Their conclusions in this area, even though based on some of the literature, are questionable and provide many grounds for disagreement. Similarly, they don't mention that most people who are exposed to the noise will be exposed indoors. Also, any one person is likely to exposed more often indoors than

outdoors. Also, why don't the authors mention the fact that the peak noise intensity falls off very rapidly as you depart from the center line of the overflight. Aircraft show a very rapid fall off of peak noise level particularly at an altitude of 250 feet where the highest peak noise levels occur. The failure to mention these factors gives a very one sided approach and suggests that they may not have a very good understanding of how the noise is propagated. Why did they not ask themselves: "What is the probability that a person will receive a noise exposure of the intensity that is directly under the flight path and what will be his noise exposure if he is 100, 1000, or 2000 feet from the center of the flight path and what will be his noise exposure if he is inside a structure when all of the noise levels occur?" Furthermore, the answer is needed to this question because some credit must be given for the time the ear has to recover, that is the whole basis of the equal energy hypothesis that there is a trade-off of time and intensity. We don't really believe that this equal energy hypothesis can be ruled out particularly if no one investigates its predictions. To do this, one must know what the noise stimulus is really like in terms of time and intensity and frequency. The authors do not know this. They indicate they do not know the true frequency for a given location much less do they know it for a given individual. To understand noise effects, one must first understand the noise stimulus. These authors apparently have an incomplete understanding of the noise stimulus, particularly in its epidemiological sense. This is a far more difficult question and procedure than generalizing from inadequately supported suppositions about level and combinations of noise frequencies that could possibly produce a damaging flyover level. The criteria proposed is not a scientific criteria and it is not a reasonable one for evaluating hearing loss. It is recognized that adverse effects to hearing may not appear for years because the damage may be insipid. That is a very hard position to argue against. Our only retort can be that such damage has not been proven by an epidemiological study conducted over the years and for all the reasons given above we believe flyover noise as it typically occurs has no hazardous effect on human hearing.

We agree with few conclusions reached by these authors in the health effects area. Their relatively uncritical review of studies and the uncritical acceptance of their own results lead them to point out "- Manifestations of long-term health effects from exposure to low altitude overflight noise can be expected in the cardiovascular system (especially increased likelihood of developing hypertension) and in the psychosomatic area." There are not enough details of procedures, methods and statistical treatment of the data to warrant agreeing with the author's conclusions. Furthermore, even with the sparse description there are enough details to know that the studies conducted and reviewed by these authors suffer from many of the same limitations as those reviewed by S. Thompson et al (85,89). The author's use of emotionally ladened words to describe effects, instead of numbers and data, detract from the effort. No where do these authors mention that in previous studies the physiological response (particularly, heart rate and blood pressure) to noise has been small. Anticipation of an event often produces larger physiological responses than does the event itself. No where do the authors mention that they controlled for anticipation. On the contrary, in their study with older people they pointed out to the subjects that in the upcoming session the flyovers may be a little louder than in the previous. No counterbalancing of condition was used in the experiment so there is strong reason to expect anticipation effects. Similarly, in their surveys with physicians, shift workers, and the elderly they used little control in interview and questionnaire procedures. They even point out that

considering the nature of the survey that it was not possible. This being true then one should only expect hypotheses for future research not "conclusions."

It is difficult to respond to an onslaught of claims of adverse effects and particularly to sensitive groups such as children and the elderly. One should keep in mind that if such groups are more sensitive than other members of the population and the evidence is not convincing that they are, it does not immediately follow that the noise under discussion is at a level that is a hazard to their health. It also, in the opinion of many, is too early to talk about the health effects of "any" type of noise, since as pointed out in "Combating Noise in the 90s" that:

> "Despite this apparently substantial body of epidemiological and animal-model evidence favoring the position that exposure to high levels of noise can raise blood pressure, a number of contemporary reviewers concluded that the evidence gathering techniques were insufficient to prove or disprove a causal relationship (9)."

In spite of claims of the biological plausibility of noise adversely affecting the cardiovascular system, there is not unequivocal evidence that it does. In those studies where noise seems to have caused the greatest increases in blood pressure, the investigators couldn't be certain about the levels of noise that produced the increases because of the difficulty in measuring the noise or only using estimates of the noise or lack of knowledge of which workers were wearing ear protectors and which ones were not. This is common in many studies of the health effects of noise that the investigator doesn't know the noise level used.. Even those that had a good estimate of the noise in the work place did not know the noise level that workers were exposed to in their leisure time. Equally as important as the incomplete knowledge of the stimulus, is the fact that most studies did not control for potentially confounding variables.

Schwartze and Thompson conclude from a review of these flyover studies that:

> "Recent studies of military low-altitude flight noise with its unusually high maximal levels and rapid rise in sound level have shown no increase in cardiovascular disease. One study of a small group of children showed high systolic blood pressures (group difference 9 mm Hg) in 9-13 year old girls, but not in boys, in the area where exposure was highest. Significantly lower heart rates were observed for boys in the low-altitude flight area as compared to controls. These could not be verified in a similar field investigation (72)."

If reviewers cannot agree that the plethora of industrial (workplace) noise studies have demonstrated significant increases in blood pressure, then there is even less reason to accept subsequent claims about the adverse effects of fly-over noise. Particularly, there is little reason to accept claims about fly-over noise producing increased mortality rates and increases in cardiovascular death, adverse effects on the learning ability of middle and low aptitude students, increase in admission to mental hospitals, and adverse effects on pregnant women and the unborn fetus.

Summary and Conclusions

1. There are probably still more authors, researchers, and experts who believe the balance of the evidence indicates **no** adverse effect of noise on the cardiovascular system. A direct

cause and effect relationship has not been demonstrated between noise exposure and adverse cardiovascular effects in any study using human subjects.

2. A cause and effect relationship between permanent hearing loss and noise exposure has been documented. Such documentation has been based on studies in which very high levels of noise have been used and, although there is still debate about this, the Leq level seems to be 90 dB or higher. Statistical studies, based on data obtained from using high levels of noise, have been used to predict "backwards" to show that some permanent hearing loss may be logically expected at $L_{eq,8hr}$ levels as low as 75 dB.

3. The EPA in their guideline and planning documents have assumed that if people are protected against "any" permanent threshold shift that they will also be protected against the possibility of permanent health effects. This agrees with the opinion expressed in several review articles (6,22,46,93). However, the next step of assuming (at least by implication) that the level (i.e. an $L_{eq,8hr}$ of 75 dB) for beginning permanent threshold shift is also the threshold for the beginning of other harmful extra-auditory effects may not be justified. However, another group of experts suggest that the threshold of noise effects for hypertension and Ischaemic heart disease may begin at an $L_{eq,8hr}$ of approximately 73 dB(A) for both aircraft noise and road traffic noise. This is not a large difference from the EPA position and considering the uncertainty of each position some compromise probably could be worked out if terms could be defined. But there is another problem, how far can the analogy between hearing loss and possible health effects (cardiovascular effects) be carried. One should keep in mind that the onset of NIPTS is evaluated by the average of the acoustic frequencies of .5, 1, 2, and 4 kHz, on an 8-hr day, for 0 to 40 years (Appendix A). This implies that level could be increased for time off from the noise source. Probably few people live in the same noise area (whether aircraft or traffic noise) for 40 years. One can readily see that it would be very difficult to come up with a logical standard. This seems like a good argument for those who say we should use those dose-response relationships that we already have for setting standards. The principle one of these is the one for Ldn and %Ha (16). In Ldn levels above 75 dB, a large percentage of the population are highly annoyed (37% +). This may be reason enough for limiting noise exposure to below that level.

4. Noise researchers have rarely considered physiological measurement and interpretation at the level of analysis required to demonstrate an effect of noise on health. Short-term physiological changes induced by noise are not a direct demonstration or a logical demonstration of the adverse effects of noise on health. Particularly, since such changes, in many studies, are smaller than would be expected from other situations (stimuli) experienced in daily living. How far this statement can be generalized is unknown since no one has systematically compared physiological changes brought about by noise and changes brought about by other types of stimuli or situations.

5. The impact of noise on the health of an individual is more likely to occur through the psychological processes of appraisals and perceived mode of coping with the noise than it is by a direct vegetative effect elicited by the autonomic nervous system (i.e., in terms of the

meaning or threat value of the noise and the perceived ability to cope with the noise). These processes occur very early and very rapidly in "response" to noise, and "intense" physiological reactions are more likely to occur because of these intervening processes than because of a direct effect of the noise itself (5). And it follows from this viewpoint that it is more appropriate to attempt to identify "noise sensitive" groups by some psychological or personality characteristic than it is to assume that a variable such as chronological age is going to determine noise sensitive groups. This is a difficult area of research, since, attempts to relate personality characteristics to **anything** have not met with remarkable success, and studies undertaken at that level often raise more questions than they answer. Conversely, there are logical reasons, though nothing has been proven, in at least some noise situations to expect that age may discriminate people in their reactions (52).

6. Noise studies that show "suggestive" adverse effects of noise on health are represented in scientific papers, planning documents, and in the popular media more than papers that show no such "suggestive" effects (i.e., they are quoted or referenced more often). This makes it difficult to communicate to the public the "real" state of the investigation into the effects of noise on people. It is made even more difficult when administrators, regardless of their motivations, make uncritical statements about the adverse effects of noise . Furthermore, many popular reviews, many in popular science magazines, are little more than a paraphrasing and quoting from administrators and scientists who have made uncritical statements about the effects of noise.

7. A study never will be conducted that can't be criticized and thus limited in its generality, and any decision about regulations that must be established, if any, should be based on our best understanding of the literature and on the weight of the evidence and not on the results of a particular study.

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Appendix A. Procedure for evaluating loss of hearing\severe health effects

In the document "Guidelines for Preparing Environmental Impact Statements on Noise " a procedure is recommended for use in evaluating Noise Induced Permanent Threshold Shifts (NIPTS) and severe health effects. The authors point out that they proceeded on the assumption that protection against noise-induced hearing loss is sufficient to protect against severe extra-auditory effects. Therefore, this procedure, to be applied only above 75 dB, is supposed to give an indication of the potential for NIPTS as well as an indicator of health effects. Although, it is none too clear how the metric is to be evaluated. The 8-hour average sound level (or the Day-Night Level (Ldn) if 16 of the 24 hours are in Leqs of 70 dB or less), is used as the input. The formula is:

$$\text{Ave NIPTS} = (L_{eq,8h} - 75)^{2/40}$$

The average NIPTS was obtained by averaging across frequency (.5kHz,2kHz,2kHz,4kHz) and time (0 to 40 years) and percentiles (.1 to .9). They also recommend presenting the values for the 90 percentile to show the predicted response of the 10% of the most sensitive population. (Technically, after a 40-year exposure.) The MAXimum NIPTS at 10 and 40 years and AVErage NIPTS are presented below:

LEVELS	MAX NIPTS(40 YR)	MAX NIPTS(10 YR)	AVE NIPTS
75	2	1	0
80	4	3	1
85	7	6	3
90	12	9	6

These levels are supported by extensive data and are analyzed in the EPA levels document. The interpretation of these results is complex. The average NIPTS, as pointed out above, is averaged across so many factors that it has little meaning except as a index (see Figure 1A). The maximum NIPTS is a little clearer. For example, the maximum NIPTS for the average of frequencies .5, 1, 2, and 4 kHz after a 40-year exposure at 90 dB is 12 dB. However, NIPTS after a 10-year exposure is 9 dB. Both NIPTS are for the 90 percentile (the top 10 % in sensitivity).

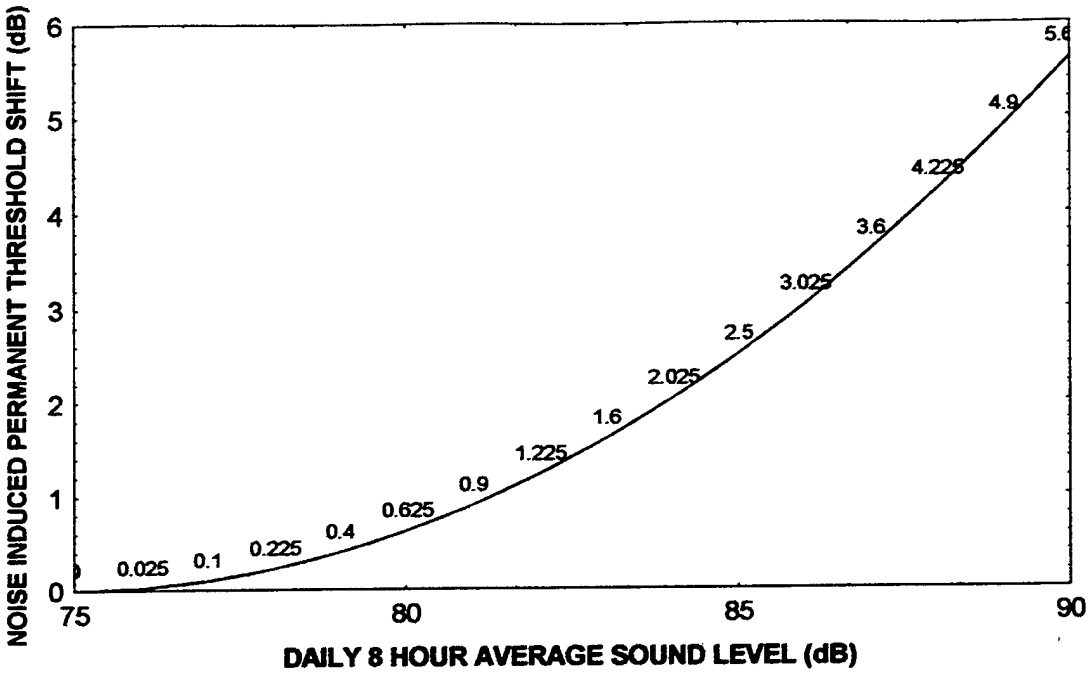


Figure 1A. Average Noise Induced Permanent Threshold Shifts.

Appendix B. Annoyance

A resurgence of interest on the effects of noise on people was brought about by the establishment of the Environmental Protection Agency (EPA) and the passage of the National Environmental Policy Act in 1970 and the Noise Control Act of 1972. These Acts established the policy of the United States Government that prior to the start of all Federally funded projects or programs that the potentially adverse impact of these actions on the environment must be assessed. The requirement for Environmental Impact Statements (EISs) with respect to noise makes it desirable that the technical criteria and evaluation procedures in the preparation of such statements are relatively uniform so that a qualitative and quantitative assessment of the adverse impact of contemplated projects and a comparison of alternative plans can be conducted.

The EPA decided in 1974 that all environmental impact assessments should use the Day-Night Average Sound Level (DNL) which is based on the A-weighted energy equivalent level penalized for night-time exposures and the Sound Exposure Level (SEL), the time-integrated, A-weighted single event measure. These decisions by EPA were a compromise of technical accuracy for a more simple index that could be easily used to measure all types of noise environments, be it aircraft, automobile traffic or industrial noise.

At the request of EPA, the National Academic of Sciences-National Research Council-Committee on Hearing, Bioacoustics, and Biomechanics (CHABA) developed and published guidelines for preparing EISs with respect to noise. The document contains guidance on the assessment of general audible noise and criteria to describe the environmental impact of vibration, infrasound, and high-energy acoustic impulses. Three tables in the document contain summaries of human effects for DNLs of 55, 65, and 75 dB. The effects are given in terms of interference with speech communication, average community reactions, community annoyance, and the importance of noise as an adverse aspect of the environment. Similarly, a dose response relationship for predicting Noise Induced Permanent Threshold Shift (NIPTS) from an eight-hour Leq is given. The authors point out that if one is protected against NIPTS, he is also protected against severe extra-auditory health effects. Therefore, it was recommended that the function can also be used for an assessment of severe health effects. Most people, preparing EISs, probably have considered this recommendation more confusing than enlightening, and haven't often used the function in their analyses. Other derivative concepts such as Sound Level Weighted Population and the Noise Impact Index seem to have been little used by the preparers of EISs. When the document was first published in 1977, it met with mixed reactions. Some were opposed to the recommended procedures, but the majority felt it brought order into chaos. Subsequently, similar guidelines have been adopted by the U. S. Air Force and Army, and various standardization groups throughout the world.

The increasing use of the A-weighted scale was based on the findings of several studies showing it to be at least as effective for associating with effects (annoyance, speech interference, and

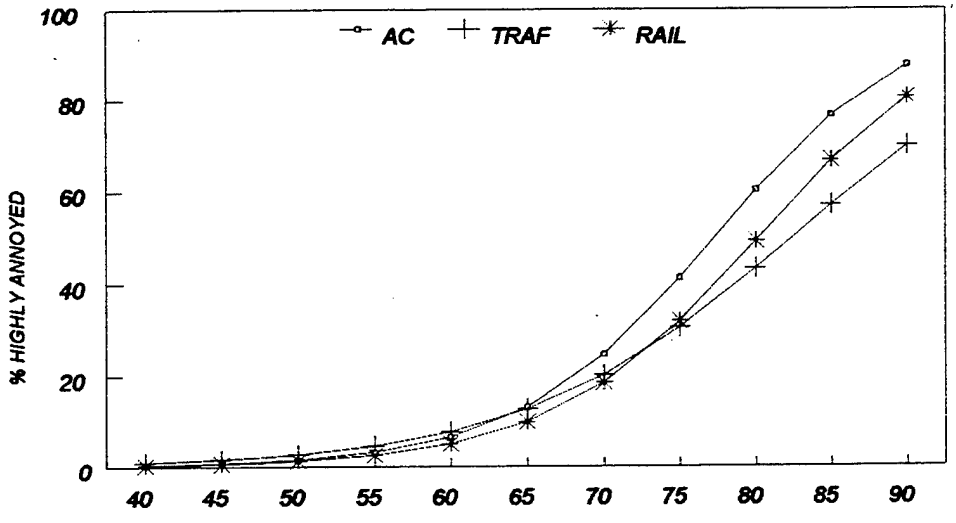
hearing loss) as more complex weightings or procedures. The A-scale weights most heavily those frequencies of greatest sensitivity to the human ear. Roughly, it gives a higher weighting for frequencies from 1000 to 6000 Hz than it does to lower and higher frequencies. An A-weighted decibel value (dBA) is used to measure noise at a given instant, to denote a maximum level, or a steady-state level. As a single number indicator of complex noise environs over longer exposure times, the Equivalent Sound Level (Leq) has been developed. The Leq may be of any duration (commonly 8-hour or 24-hour) and sums all of the time-varying A-weighted sound energy in the noise environment, regardless of source. To say that a complex sound environment has an Leq of 60 db is to say that the sound contained in that environment is equivalent to a continuous sound of 60 dB as measured on the A-scale for the specified time period. The most common measure used to characterize sound levels in residential communities is the DNL, which is the 24-hour A-weighted Leq with 10 decibels added to night-time sounds (10 PM- 7 AM). The arrival at the use of the DNL is not a small feat and it must be said that, worldwide, there are still a multiplicity of noise rating schemes in use; however, it is getting better and there is a definite trend toward a greater acceptance of the A-weighted scale and Leq type noise measures.

What measures do we use to measure the effects of noise on people? Of course, the diversity of proposed measures of effects are legend. Before and during the 1950's, the effects were determined largely from anecdotal evidence, case studies, laboratory studies, and a very few social surveys. Even in these early days, "annoyance" was given prominence. How annoyed people were as a consequence of the noise exposure was thought to be very important, and indeed, that is the belief to the present day. Also, in more recent years, there has been more emphasis on obtaining data on community response from social surveys of the communities involved. There has been a great effort directed toward finding a relationship between the noise exposure metric and some measure of activity interference (assumed by most researchers to be primarily communication interference) or annoyance as measured by a social survey. A wide variety of responses have been used in social surveys in an attempt to determine intrusiveness, disturbance of speech communications or sleep, interference with TV or radio listening and interference with outdoor living. The overall response to all of these factors was measured by questions on the annoyance reaction. The concept of "percent highly annoyed" in the sampled populations seemed to provide the most consistent response of a community to a particular noise environment. In an attempt to meet the demand for a usable and uniform relationship, Schultz (1) reviewed the results of a number of social surveys where data were available to make a consistent judgment concerning what percent of the population was "highly annoyed" (%HA). The surveys were of community reactions to several types of transportation noises such as road traffic, railroad and aircraft noises. The results agreed fairly well with one another and he developed an equation for describing the relationship between the level of exposure in DNL with the %HA. This relationship was adopted by CHABA Working Group 69 in the guidelines discussed before and subsequently, it was proposed by EPA as the appropriate function to use for the evaluation of the effects of noise on communities. Many of the early disagreements have been resolved. Suffice it to say that there can be little question that for aircraft noise the relationship has held up and given reliable results in several subsequent studies. Fidell et al (2) have updated the data base originally used by Schultz (1) for deriving the relationship between

the DNL and %HA. These authors added 239 data points to the original 161 date points analyzed by Schultz (1) for a total of 400 points. The Air Force uses a Logistics type regression curve fit for describing the relationship between DNL and %HA because it gives essentially the same predictive values as the function recommended in the Guidelines article and has the further advantage that no predictions of less than 0% or more that 100% can be obtained. Logistic fits to the 161 data points and to the 400 data points give such similar predictive values (within about 1%) that no advantage would be obtained by replacing the original Logistic equation for describing the relationship.

One question that still persists is whether the DNL-%HA relationship is the same for all types of transportation noise. Of the 400 data points, 173 were for aircraft noise, 170 for traffic noise and 57 for railway noise. In Figure 1B, plots are given for logistic fits to each of these three sets of data points. Although values for traffic and railway noise are not as high as the values predicted for aircraft noise at the higher DNL values, there are no statistically significant differences between predicted value at any DNL value.

COMPARISON OF LOGISTIC FITS
TO AIRCRAFT, TRAFFIC AND RAIL DATA

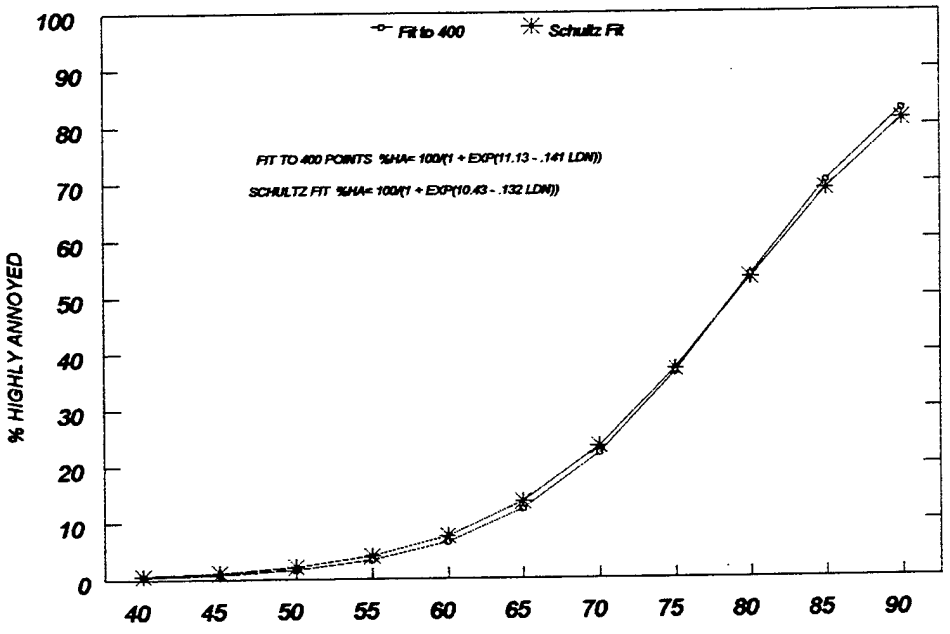


AC	0.326	0.7	1.5	3.17	6.58	13.17	24.64	41.32	60.26	78.56	87.56
TRAF	0.906	1.57	2.69	4.59	7.722	12.79	20.18	30.52	43.34	57.03	69.76
RAIL	0.29	0.6	1.23	2.51	5.05	9.91	18.52	31.96	49.25	66.73	80.58

DAY - NIGHT AVERAGE SOUND LEVEL

Figure 1B. Comparison of Air, Rail & Road Traffic (derived from Fidell et al (2)).

Time-averaged methodologies are best used to predict long-term responses to noise. Annoyance in the context of Ldn methodology is a description of a person's reaction to their noise environment after they have been exposed to it for a long period of time -- at the very least two or three months. Thus, this methodology cannot predict immediate response to a single aircraft flyover, or initial reactions to a dramatically different noise exposure scenario. Similarly, sometimes the methodology doesn't seem to do very well at predicting annoyance effects at low DNL levels. Often there is good reason for this, the community is simply not reacting to noise intensity. The DNL - %HA is the relationship between noise intensity and the percent highly annoyed; therefore, one should not expect the equation to predict those situations where noise no longer is serving as a measure of intensity, but is in fact a surrogate for many other social and psychological variables. These situations are indeed very difficult if not impossible to predict, however, this may or may not be a weakness of Ldn methodology. The Ldn - %HA curve used by the U.S. Air Force can be seen below.



Fit to 400	0.41	0.831	1.66	3.31	6.48	12.29	22.1	36.47	53.74	70.16	82.64
Schultz Fit	0.576	1.11	2.12	4.03	7.52	13.59	23.32	37.05	53.25	68.78	81

DAY - NIGHT AVERAGE SOUND LEVEL

Figure 2B. Comparison of fit to Schultz data with fit to 400 data points (2).

Despite these limitations, this general DNL methodology has the broad support of a scientific consensus and can be used to compare long-term changes in the noise environment. While individual or anecdotal testimony may deviate from the average responses predicted from careful experiments or social surveys, the DNL methodology has consistently been shown in the national

and international literature to work for large numbers of people under a wide-range of noise conditions (including loud and soft noise levels, and frequent and infrequent numbers of discrete aircraft events). There is simply no comparable approach.

REFERENCES

1. Schultz, T.J. Synthesis of Social Surveys on Noise Annoyance, J.Acoust.Soc.Am., pp 18-27, 1978.

2. Fidell, S., Barber, D., and Schultz, T. J. "Updating a Dosage-Effect Relationship for the Prevalence of Annoyance Due to General Transportation Noise." HSD-TR-89-009, U.S. Air Force, Noise and Sonic Boom Impact Technology, Wright-Patterson AFB, Ohio, December, 1989.